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Archives of Neurology and Psychiatry

Vol. 11 JANUARY, 1924 No. 1

A STUDY OF TUMORS ARISING FROM EPENDYMAL CELLS*

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It is evident from an attentive reading of the descriptions of tumors called "ependymal gliomas" that the vast majority of them did not originate from ependymal cells. This fact has been noted by many others. Indeed, the tumors seem often to have been thus designated for no better reason that that they lay mostly in the ventricles of the brain or protruded markedly into them.

The nomenclature of tumors is, for the most part, taken from the tissues whence they are presumed to arise or, to be more exact, whose structure they most accurately reproduce. We may mention four criteria used to determine this point: (1) direct observation of the transition of normal tissue cells into tumor cells; (2) the architecture of the tumor, that is, the relationship of the cells to each other and to the surrounding structures; (3) the internal structure of the cells and their intercellular excretions; and (4) specific microphysicochemical reactions of the cell cytoplasm.

Usually a microscopic preparation is necessary for the application of these criteria. Observation of the transition of normal into tumor cells is rarely possible. The relationship of the tumor cells to each other and to the surrounding structures is in most cases sufficiently evident if the nuclei are brought out distinctly. The pathologist is also interested in the possible presence of mitotic figures, hence the usual laboratory procedure of staining with hematoxylin and eosin on material fixed in Zenker's fluid, which maintains well the structural relations, and permits at the same time an acceptable nuclear stain. Often, however, the structure of the tumor is too atypical to allow one to arrive at a diagnosis by this simple means. It is then necessary to study the internal structure of the cells and their intercellular excretions. The possibilities of this method of approach are evident from a perusal of

* From the Surgical Clinic of Dr. Harvey Cushing.

* Read before the annual meeting of the American Neurological Association, Boston, May 31, 1923.

Mallory's work on pathologic histology. It is also necessary in some cases to distinguish between cells whose internal morphology offers no characteristic distinguishing features and which produce no known intercellular excretion. For example, the supporting tissue of the brain by ordinary histologic methods shows nothing remarkable, yet the microphysicochemical methods of the Cajal school of neurohistologists in Spain have revealed an amazing differentiation in this tissue. The method of attack by utilizing reactions of the cytoplasm of the cells has begun to find extension outside the nervous system under the direction of del Río-Hortega.[1]

In the case of the ependymal cells, if one is not fortunate enough to observe the transition into tumor cells, one must depend on the internal morphology of the cells and their relationship to surrounding tissues to determine the diagnosis of tumors arising from them, for there is no specific microphysicochemical reaction by which they may be recognized and they produce no known intercellular excretion. Luckily, both the internal morphology of ependymal cells and their relation to surrounding structures are rather characteristic.

STRUCTURE OF NORMAL EPENDYMA

The application of any of the foregoing criteria demands a knowledge of normal histology, and as the structure of the normal ependyma is not well known it may be wise to recall here a few essential facts. They are mostly taken from the study by Studnicka,[2] which I have never seen mentioned in any article on "ependymal gliomas." It deals mostly with lower vertebrates. The structure of the human ependyma is very imperfectly known.

Of the various types into which the cells of the neural tube differentiate, there are two which come to line the ventricles of the brain and differentiate from each other rather late, the ependymal and the choroid cells. The ependymal cells lie over the massive portions of the brain, adjacent to a dense layer of fibrillary glia known as the subependymal glia. The choroid cells lie on connective tissue and show a marked tendency to wrap themselves around blood vessels and to form tufts. Their internal morphology is quite different from that of the ependymal cells.

There are also certain areas in the lining of the ventricles formed by a flat layer of epithelial cells, in some places accompanied by a thin layer of glia tissue, which show no tendency to wrap themselves around the blood vessels of the underlying connective tissue to form tufts.

1. Del Río-Hortega, P.: Arch. d. cardiol. y hematol. **2**:161, 1921.
2. Studnicka: Anat. Hefte, Abt. 1 **15**:301, 1900.

These are known as the velar areas, of which the velum medullare posterius is a good example. The cells are cuboidal or somewhat flattened and have the same internal morphology as the ependymal cells.

The fundamental difference in structural relationship of the choroid and ependymal cells is easily understood from the course of histogenesis of the nervous system. The neural plate is primitively a simple epithelium, with the cells facing on an internal and an external limiting membrane. Between these limiting membranes occurs an extraordinary proliferation of cells, which form the mass of the central nervous system. There are certain areas, however, in which this proliferation does not occur, namely, the choroid and velar areas, and here there remains even in the adult a simple epithelium.

The subpial and ventricular surfaces of the brain are therefore analogous to the internal and external limiting membranes of the choroid and velar epithelium. The ependymal cells originally faced, at their other extremity, on the subpial surface of the brain, but by the multiplication of elements among themselves, and between the limiting membranes, they became first elongated and distorted until most of them finally lost their foothold on the subpial surface as it was pushed far away from the ventricular surface. For a considerable period in embryologic development this double attachment persisted. In certain places where the neural tube remains relatively thin, this primitive structure exists even in the adult, as has been demonstrated by the Golgi method in the hands of von Kölliker, van Lenhossék, Nansen, Cajal, Retzius and others. In the adult, also, many of the ependymal cells have processes extending into the depths of the nervous tissue, clearly seen in Heidenhain preparations, which do not extend so far as the subpial surface, but most of them have lost all relationship with the internal limiting membrane and form merely a lining epithelium for the ventricles.

The shape of the ependymal cells varies greatly in different parts of the nervous system, from flattened types, rectangular or spindle-shaped on cross section, to cuboidal or even cylindrical. They are always higher over the blood vessels than over the solid brain wall. They are particularly well developed in the rhomboid fossa, the aqueduct of Sylvius and the central canal of the spinal cord. Over the greater part of the brain wall a new internal limiting membrane has been reconstructed.

The processes of the ependymal cells were first seen by Hannover. There is, according to Ziehen, usually only one to each cell, but it may be branched. They do not stain typically by methods used for the demonstration of neuroglia fibrils.

The ependymal cells of the human embryo, in common with those of the lower vertebrates, are ciliated, and at the base of each cilium is a small body known to the German investigators as "Basalkörperchen" or "Blepharoplasten" (Fig. 1). It may be a single granule, a pair of

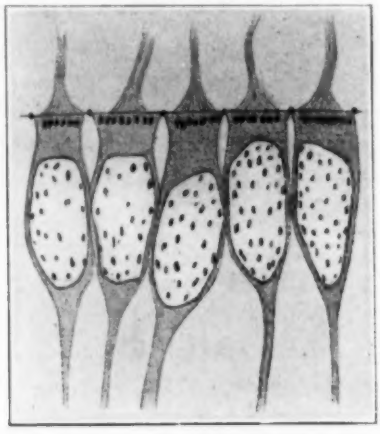

Fig. 1.—Copied from Studnicka. Ependymal cells from the rhomboid fossa of a human fetus showing blepharoplasten.

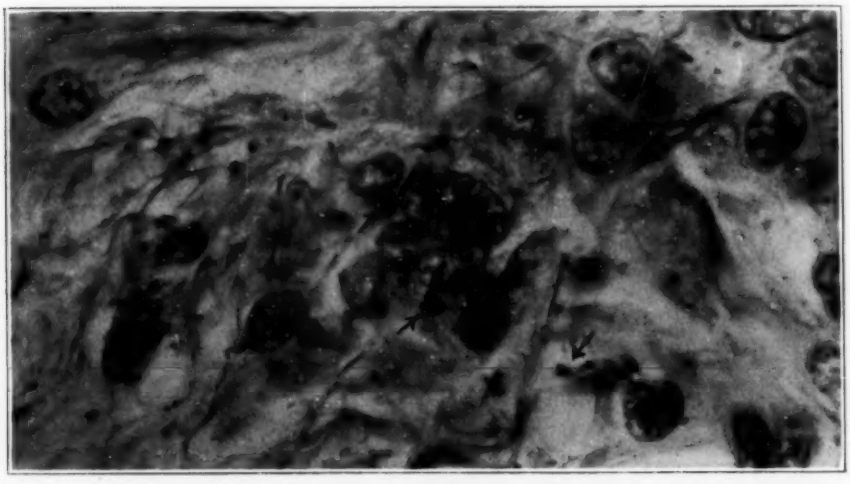

Fig. 2.—Ependymal cells from the region of the central canal of the spinal cord in a case of subacute combined cord degeneration. Blepharoplasten are indicated by arrows. Neutral ethyl violet-orange G stain; × 1,200.

granules lying one above the other, a minute bacillus-like rod, or in the form of a dumbbell. For a short period in embryonic life blepharoplasten are also seen in the choroid cells of the lateral ventricles. They

are only transitorily present in most of the ependymal cells, but persist in the adult in the cells of the aqueduct of Sylvius, the rhomboid fossa and the central canal of the spinal cord (Fig. 2). They lie deep under the surface of the cells, and remain even after the cilia have disappeared.

Blepharoplasten were seen in the ependymal cells by Marchi and were well described by von Engelmann. Weigert, apparently ignorant of previous work, described them anew in his monograph on the neuroglia. Mallory used them as an aid in establishing the ependymal origin of cells in tumors. They stain by any method for the demonstration of neuroglia fibrils. Their nature is unknown, although they are supposed by some to be akin to centrosomes.[3] Similar bodies are found at the bases of the cilia of the cells of the epididymis, and in other ciliated cells.

It is known that even in postfetal life neuroglia cells are formed from the ependyma. There is some evidence that this formation is accelerated by pathologic processes. In certain cases of ependymitis, del Río-Hortega [4] has been able to follow the transformation of ependymal cells into neuroglia cells by the changes in morphology and reduction in the number of blepharoplasten. Near the ventricles in cases of cerebral thrombosis, also, he has found cells with groups of typical blepharoplasten, which he believes to be emigrated ependymal cells (Fig. 3).

LITERATURE

From the literature of so-called "ependymal gliomas," it is at once evident that Mallory is practically alone in applying a knowledge of the normal microscopic structure of ependymal cells to their recognition in tumors of the central nervous system.

Tumors recorded as "ependymal gliomas" seem to be of four types:

1. Gliomas arising from the subependymal region. Their number is legion as recorded by Virchow, Bielschowsky, Pfeifer, Brüning, Hunziker, Christin and Naville, and others. The ependyma may continue in an unbroken layer over the surface of these tumors, or be completely absent as in the case of the granulations of ependymitis granularis.

2. In the midst of certain gliomas of the hemispheres, cysts of greater or less size may be found lined with cells which resemble superficially those of the ependyma. Such instances have been recorded by

---

3. Nerve cells, contrary to the common opinion, contain a diplosome in the periphery of the cytoplasm, which can be demonstrated by special methods (del Río-Hortega, Trab. del lab. de invest. biol. de la Univ. de Madrid **14**:117, 1916). The centrosome of the neuroglia cells is a single heavy, spherical body in the cytoplasm near the nucleus (Cajal, Ibid. **11**:255, 1913).

4. Del Río-Hortega, P.: Trab. d. lab. d. invest. biol. d. l. Univ. de Madrid **14**:1, 1916.

Stroebe, Saxer, Bücholz, Bonome, Henneberg, Herzog, and others. Similar formations were found in the spinal cord by Rosenthal, Spiller, Marburg and others.

3. Ventricular papillomas consisting of a core of subependymal glia covered by a more or less proliferated ependyma. A beautiful example has been described by Linck.[5]

A workman, aged 44, died suddenly while being anesthetized with chloroform. During the necropsy a pea-sized tumor was unexpectedly found in the floor of the fourth ventricle just in front of the calamus scriptorius. The tumor was a

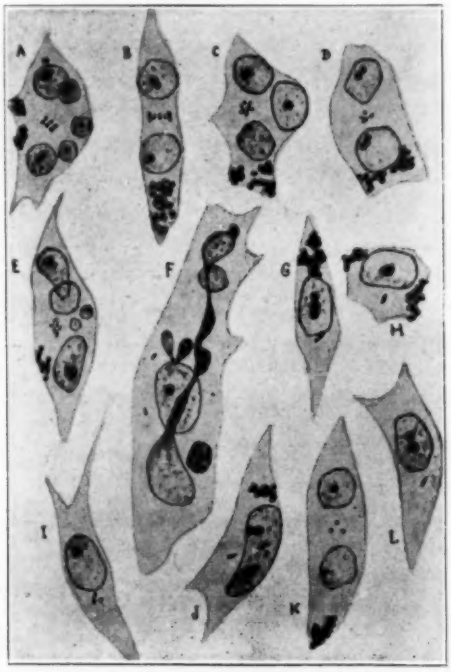

Fig. 3.—Copied from del Río-Hortega. Cells from the ventricular border of a small focus of subependymal softening. Blepharoplasten clearly shown as well as coarser granules of a different nature.

papillomatous growth attached by a broad base to the medulla oblongata. Microscopically it was formed of a core of very fibrous neuroglia, an extension of the subependymal layer, covered by ependymal cells. The ependymal layer was usually composed of a single row of cuboidal cells, but in many places it was three or even four cells in thickness. The tumor contained no connective tissue except in the walls of very rare blood vessels lying in the larger ramifications of the growth. Nothing was said of the internal structure of the ependymal cells.

5. Linck: Ziegler's Beiträge **33**:98, 1903.

4. True ependymomas—tumors originating from ependymal cells and more or less closely reproducing their structure. They are very rare and I have identified in the literature only five cases which seem to me authentic.

Muthmann and Sauerbeck,[6] in an excellent study, established the origin of a tumor of the fourth ventricle by means of serial sections.

A man, aged 22, came under observation complaining of headache, vertigo and failing vision. From the age of 8 or 10 years he had had headaches, usually in the morning. He died after an exploratory operation and at necropsy a tumor about 4.3 cm. long by 3.2 cm. in width was found in the fourth ventricle. It was firm, reddish gray and somewhat translucent. There was but one point of attachment, to the velum medullare posterius; otherwise it lay free in the ventricle.

The growth was fairly vascular, with wide blood channels here and there, but no capillaries. The greater part of the tumor was made up of elongated cells forming a many layered epithelium, one end of the cells being drawn out into a long process extending into the depth of the tumor and usually ending on the wall of a blood vessel. Large areas were made up of a dense mass of similar cells, coming off from which no processes could be seen, probably because they were cut in cross-section.

Nothing resembling neuroglia fibers was found, and nothing is said of the internal structure of the cells. The tumor was definitely attached to the velum medullare posterius, a little stalk projecting into it, but a transition from normal to tumor cells was not clearly seen.

Spiller[7] has described a remarkable case which seems unquestionably to be a tumor arising from the ependyma of the fourth ventricle.

The growth "filled the posterior part of the fourth ventricle and extended downward upon the cord as low as the sixth cervical segment. This tumor also contained rows of cells resembling those of the ependyma, and forming the lining of large, irregular spaces. It was an ependymoma. Another, but smaller tumor, having the same structure, was found upon the lower part of the thoracic region of the cord and was probably a metastatic growth, and therefore exceedingly interesting."

No mention is made of neuroglia fibers and nothing is said of the internal structure of the cells, but having had the opportunity, through the kindness of Dr. Spiller, to examine the sections, I am convinced that the tumor arose from the ependymal cells in the floor of the fourth ventricle.

Mallory,[8] in an exceedingly important article which seems to be generally overlooked by foreign writers, in three of his cases used the internal structure of the cells to establish the origin of the tumors from ependymal cells. Two from the fourth ventricle will be summarized briefly. The third was an exceedingly rare growth in the sacral region.

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6. Muthmann und Sauerbeck: Ziegler's Beiträge **34**:445, 1903.
7. Spiller, W. G.: J. Nerv. & Ment. Dis. **34**:297, 1907.
8. Mallory: J. Res. **3**:1, 1902.

CASE 1.—The patient was an adult, but no clinical details are given. Pendant from the roof of the fourth ventricle was an irregularly spherical tumor, 4 by 5 by 3 cm. in size, reddish gray in color, soft in consistency, and very vascular. Microscopically, the tumor consisted of two different structures; of cells which formed a fairly coarse meshwork, and of large blood vessels, surrounded by connective tissue, which lay in the spaces of the meshwork. The cells were distinguished by two peculiarities: most of them contained in their protoplasm the markings characteristic of ependymal cells and they had running between them and touching their protoplasm numerous delicate neuroglia fibers. The cells were epithelial in type. In this mass of protoplasm there were often found very small, round or irregular, sharply defined cavities like the lumen of a small gland. These cavities were lined by cells exactly like the ependymal cells. Like them, they also contained the characteristic markings in the protoplasm on the side adjoining the lumen. In sections stained by the phosphotungstic acid-hematin method, the markings were found to be almost invariably in a more lightly staining area of the protoplasm. They showed a tendency to arrange themselves around the periphery of this lighter area. They varied in number from about ten to thirty. In shape the markings varied from round and oval bodies to definite rods with square ends, which were from two to four times as long as broad.

CASE 2.—The patient was a boy, aged 17, who had had symptoms for over two years indicative of internal hydrocephalus. There was a tumor in the fourth ventricle which it completely filled. It formed a semitranslucent grayish, somewhat pear-shaped mass, measuring 5 by 6.5 by 3 cm. It was attached to the floor of the ventricle and the smaller conical end projected beneath the pia over the posterior surface of the pons and the beginning of the medulla. On section it presented the same grayish translucent appearance as the outer surface. Microscopically the tumor was composed of areas consisting chiefly of neuroglia fibers and of other areas of about equal extent, which were very cellular. The two forms of tissue were not very sharply marked off from each other, but tended to fuse together. The cellular areas closely resembled the appearance of the first tumor described. The cells tended to arrange themselves in clumps; occasionally there occurred well-defined cavities lined by definite epithelium. In this tumor, as in the other, small groups of the characteristic ependymal cell markings were present in the protoplasm of the cells. They were not so numerous as in the first case, but varied from two to about ten in number in a cell. As a rule they were round or oval, but occasionally rod-shaped.

PERSONAL OBSERVATIONS

In a series of 868 consecutive verified intracranial tumors, observed in the clinic of Dr. Harvey Cushing, I have been able to find only six which seem to me to have arisen from ependymal cells, four from the ependymal floor of the fourth ventricle, and two from the velum medullare posterius (tela chorioidea ventriculi quarti). As only one specimen was obtained at necropsy, the diagnoses are based on the gross findings at operation and on the histologic examination of material removed at the time of operation.

In the one case in which a necropsy was obtained, the brain was fixed in 10 per cent. solution of liquor formaldehydi; in all other

instances the operative material was fixed immediately in Zenker's solution. Paraffin sections cut at 5 microns were stained with hematoxylin and eosin, methylene blue and eosin, Mallory's connective tissue stain, van Giesen's method, Mallory's phosphotungstic acid-hematoxylin, and finally by the neutral ethyl violet-orange G and acid fuchsin-methyl green methods for neuroglia fibrils, which I have described elsewhere.[9]

The case reports will be divided into two groups to accord with the presumed origin of the tumors.

A. EPENDYMOMAS OF THE RHOMBOID FOSSA

These tumors were in every case firmly attached to the floor of the fourth ventricle. In the first no material was obtained for examination, but it is included because of its great clinical interest.

CASE 1.—*Ependymoma (?) of the fourth ventricle, unverified. Operation with recovery and survival after eleven years.*

G. MacP., aged 25, a housewife, was admitted Feb. 11, 1912, complaining of occipital pain, stiffness of the muscles of the neck, staggering, failing vision and vomiting, having been referred by Dr. Turnbull of Winnipeg, Manitoba. The illness began in February, 1911, with a "sinking feeling," usually noticed at night. Soon in walking there was an inclination to stagger to the left. This difficulty steadily progressed. About this same time a roaring noise began in the left ear, at first intermittent, soon continuous. In March, 1911, headaches began, at first frontal but later occipital or suboccipital and practically constant. Her condition remained about the same until May, 1911, when she became pregnant. The onset of vomiting seems to have been associated with the pregnancy, for at first it was always accompanied by nausea, was independent of the headaches and occurred in the morning. In October, 1911, she had a spontaneous miscarriage. At about the same time visual disturbances were noted in the form of flashes of light dancing before the eyes, distortion of the image, etc., and visual acuity began gradually to fail. In January, 1912, appeared stiffness in the muscles of the back of the neck, which was worse during severe attacks of headache, when the head would be held flexed and could not be extended without great increase of pain. There had never been any difficulty of speech nor of deglutition.

Positive Neurologic Findings.—The corneal reflexes were sluggish, more so on the left side; there were a few nystagmoid jerks on looking to the left; gait and station were very uncertain, with a tendency to deviate and fall toward the left; there was suboccipital tenderness and stiffness of the neck muscles; the optic disks were choked, the right being elevated 6 diopters and the left 4 diopters. In addition there was a purulent discharge from the left ear.

Operation.—Feb. 19, 1912, Dr. Cushing exposed the cerebellum without difficulty and it bulged markedly through the dural opening. Puncture of the lateral ventricle reduced the tension and allowed a complete exposure. The posterior half of the atlas was removed and the dural incision carried down to the axis, exposing the tumor, which lay between the tonsillae cerebelli and extended down into the spinal canal (Fig. 4). No attempt was made to extirpate the tumor and without removing a fragment for study the wound was closed in layers as usual.

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9. Bailey, Percival: J. M. Res. **44**:73, 1923.

Postoperative Course.—Convalescence was uninterrupted and the patient was discharged March 10, 1912, with subjective discomforts completely relieved. Gait was slightly unsteady and visual acuity was much diminished, although better than on admission. The optic disks were still elevated 3 diopters.

Jan. 31, 1914, she reported that she still had to be a little careful when coming down stairs, but was otherwise well.

July 26, 1921, she wrote that she was "as well as mortal could possibly be," doing all her housework, and the mother of four healthy children.

CASE 2.—*Ependymoma of the fourth ventricle. Operation, followed by recovery. Death seven months later.*

A. B., aged 34, a grocer, was admitted Oct. 3, 1920, complaining of dizziness and loss of vision, having been referred by Dr. A. D. Dyas of St. Stephen,

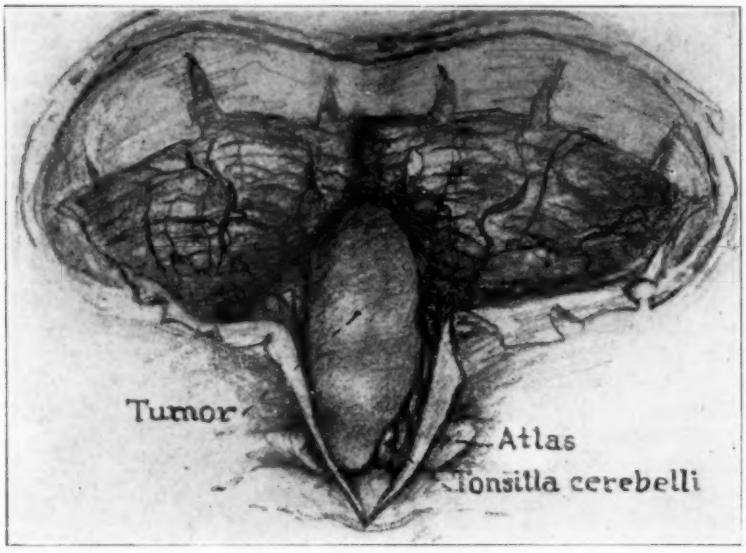

Fig. 4.—Gross appearance at operation of tumor of Case 1.

New Brunswick. In May, 1919, he began to have slight staggering when walking, followed shortly afterward by attacks of projectile vomiting, with slight nausea. From August, 1919, the symptoms gradually subsided and the patient was practically well until May, 1920, when the previously noted symptoms returned, this time accompanied by severe headache. Soon his eyesight began to fail. One week before admission he fell unconscious and was afterward mentally confused. There seem to have been no convulsive movements. There had been two similar attacks previously with unconsciousness during several minutes.

Positive Neurologic Findings.—The findings were: Bilateral choked disk, with from 4 to 5 diopters of swelling; staggering gait, with a tendency to deviate to the left; equilibrium unstable with a tendency to fall to the left and backward; ataxia of cerebellar type of all extremities; nystagmus on looking to the left and right; mental dulness; yawning and nose rubbing pronounced; persistent hiccough.

Operation.—Oct. 15, 1920, Dr. Cushing performed a suboccipital exploration with partial removal of tumor in the fourth ventricle. The patient took the anesthetic badly, so that the ventricle was punctured early. A large amount of fluid was secured, after which respiration was regular and free, and the operation was continued without difficulty. The bone was very thin. On opening the dura, the hemispheres protruded somewhat. Punctures of the hemispheres revealed no lesion. Only after thorough exposure with division of the dura down through the region of the foramen magnum was it apparent that there was a tumor occupying the posterior cistern and protruding between the two lips of the cerebellum (Fig. 5).

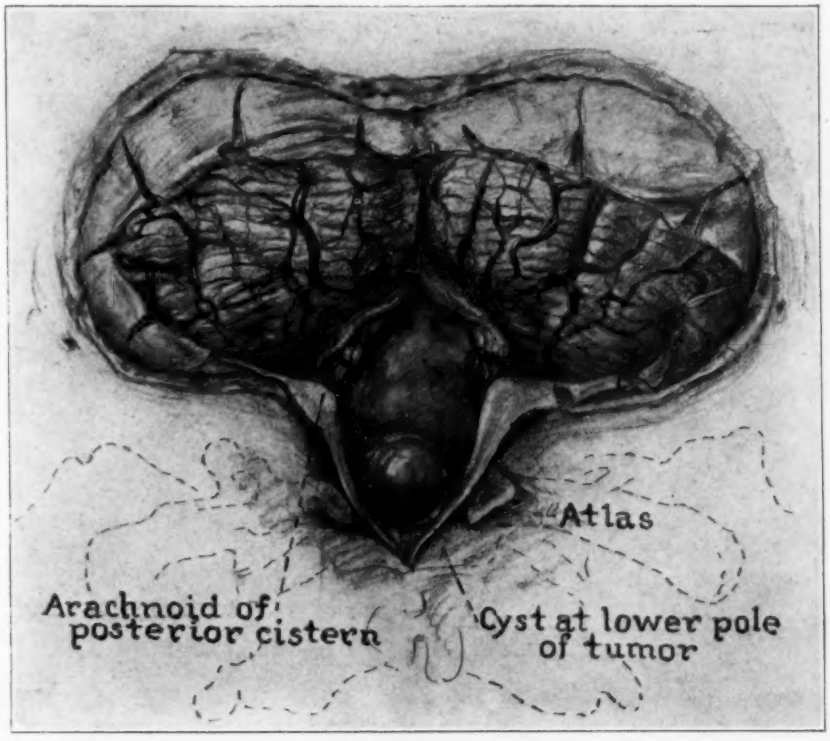

Fig. 5.—Drawing showing appearance at operation of tumor of Case 2.

On opening the arachnoid cistern a growth was seen in the midline projecting down into the spinal canal. The posterior arch of the atlas was then removed. The dura was incised down to the top of the axis, leaving a good view of the lower pole of the tumor and the spinal cord below it. This lower pole contained a cyst, which was evacuated. The wall of the tumor and the cyst were fixed with Zenker's fluid and steps were taken to begin enucleation from below upwards. It was soon found, however, that the tumor was attached to and apparently grew from the floor of the fourth ventricle, and manipulation caused respiratory embarrassment.

The best that could be done, therefore, was to expose the tumor as high as possible and split it in the midline. The tumor was of a dark reddish-brown

color and of soft consistency. Two fairly large fragments were removed with a pituitary spoon. Bleeding was controlled with Zenker's fluid. The wound was closed as usual, without a drain.

Postoperative Course.—Headaches were relieved and the mental condition returned to normal. The patient became able to walk with the aid of an orderly. Occasional vomiting persisted, without nausea. He was given two roentgen-ray treatments, and was discharged Nov. 29, 1920.

He remained at home entirely free from headaches, able to read, and able to walk with assistance until three days before death, when he complained of severe headache, became stuporous and died April 30, 1921. There was no necropsy.

Microscopic Examination.—The tumor is composed of an almost solid mosaic of polygonal cells jammed against each other, with sharply defined cell boun-

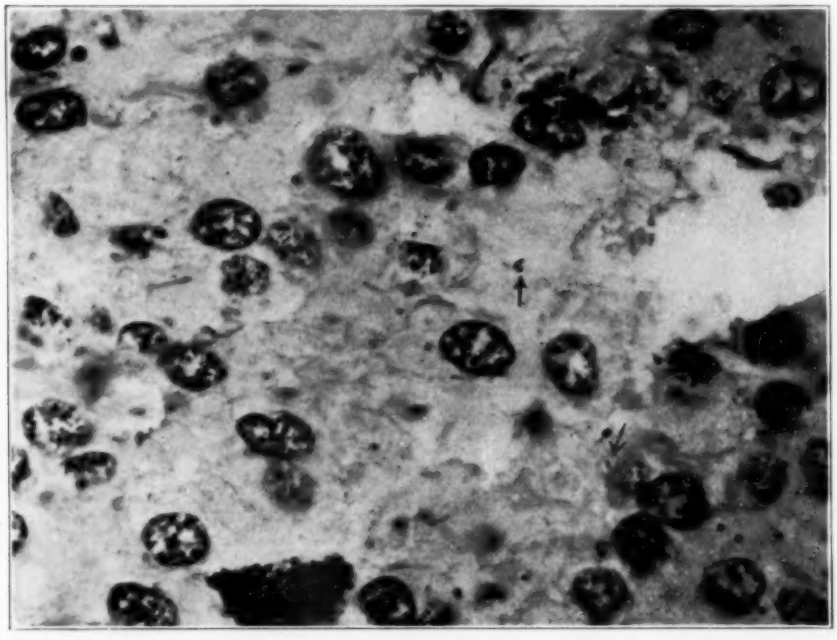

Fig. 6.—Photomicrograph of tumor from Case 2. Blepharoplasten are indicated by arrows. Neutral ethyl violet-orange G stain; × 850.

daries. These cells have no particular architectural arrangement. Sometimes an ill defined tendency to arrange themselves radially around vessels may be seen. The blood vessels are rare and have thin walls containing a few fine fibrils of collagen.

Running between the cells are scanty coarse fibrils which stain with neuroglia fibril stains, though not very sharply. The cells themselves have heavy granular cytoplasm and large vesicular nuclei.

The ethyl violet-orange G preparations were the most instructive. There might be seen in almost every cell, sometimes one, but usually two, heavily stained round markings (Fig. 6). They lay side by side, surrounded by a clear halo, looking like a diplococcus with its capsule unstained. Often three in a

group, occasionally several, would be found. Rarely, cells packed with heavy globules were present, such as were seen more frequently in the tumor of Case 3.

CASE 3.—*Ependymoma of the fourth ventricle. Operation, fatal ending. No necropsy.*

J. K., aged 7, a schoolboy, was admitted Sept. 14, 1922, complaining of headache and vomiting, having been referred by Dr. Lewis W. Hill of Boston. During the latter part of June, 1922, he began to vomit and since then vomiting had occurred almost every day. About the middle of August headaches com-

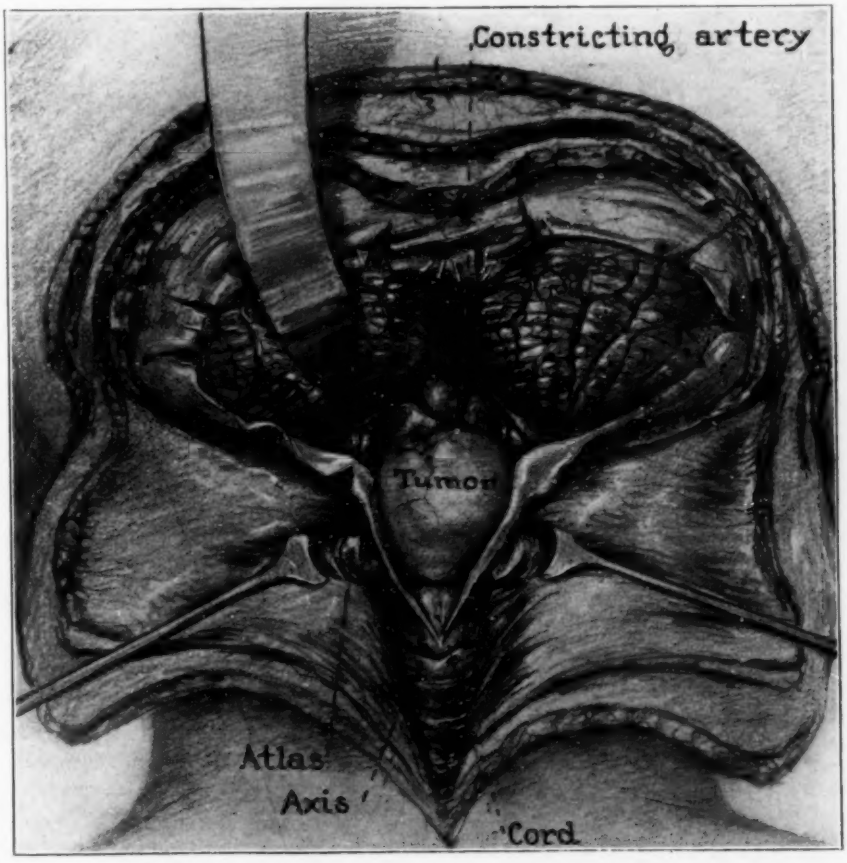

Fig. 7.—Drawing showing gross appearance at time of operation in Case 3.

menced, mostly frontal and very severe. About a week before admission he complained of double vision, and became somewhat unsteady in walking. In the last few days he complained of blurring of vision and a feeling of stiffness in the left hand.

Positive Neurologic Findings.—A cracked-pot note was elicited on percussion of the skull; suboccipital tenderness was present and was more marked on the left; there were bilateral choked disks with swelling of 4 diopters; nystagmus occurred on looking to the right or left, and was coarser to the left; gait

and station were unsteady; incoordination of cerebellar type affected all the extremities, but was more evident on the left side.

Operation.—Sept. 18, 1922, Dr. Cushing performed a suboccipital exploration with partial extirpation of the tumor. This was a simple, straightforward exploration. After puncturing the ventricle, the hemispheres were uncovered by opening the dura. The hemispheres appeared normal, but on opening the posterior cistern the tumor was seen in the fourth ventricle. The laminae of the atlas were removed and the dura was incised down to the axis. It was then possible to go below the tumor and tilt it upward. Across its posterior surface ran two constricting arteries (Fig. 7), which were divided and stripped to the side. An attempt was made to separate the tumor from the floor of the fourth

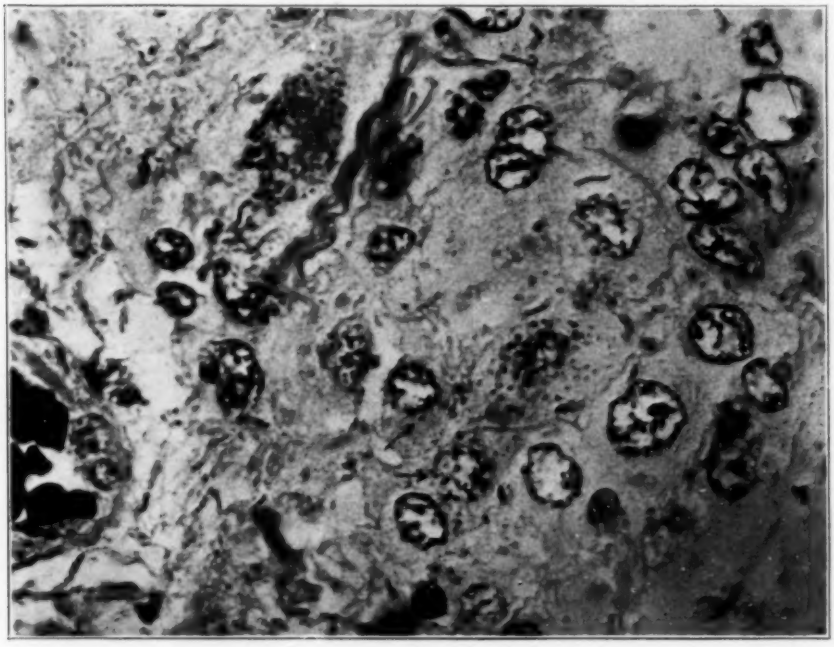

Fig. 8.—Photomicrograph of tumor in Case 3. Blepharoplasten are indicated by arrows. Neutral ethyl violet-orange G stain; × 850.

ventricle, but the separation left a bleeding surface and disturbed respiration, which on two occasions almost stopped. The portion which had been separated was removed, and the wound was closed.

Postoperative Course.—Temperature rose steadily. At 8 a. m., September 19, it was 105 and remained between 104 and 106. At 9:30 a. m., lumbar puncture was done, following which the patient had great respiratory embarrassment until a ventricular puncture was made. He did very well during the day, but began to have respiratory difficulties again toward evening, gradually became comatose, and died September 21. Necropsy was refused.

Microscopic Examination.—Microscopically the tumor consists for the most part of a mass of polygonal cells with well-defined cell boundaries, pressed closely together without any definite architectural arrangement. In other places

the structure is looser. Here the cells are pear-shaped and have a tendency to arrange themselves roughly around the blood vessels.

The character of the denser part is shown in Figure 8. The cells form a mosaic. They have large vesicular nuclei and abundant, heavy, granular cytoplasm. In almost every cell may be seen markings varying in number from two to twelve or more. The clearest pictures are seen with ethyl violet-orange G sections. Here these markings appear as heavily blue-stained granules of various sizes, often with a clear halo around them. Some of the cells contain numerous, much larger granules, which stain more feebly and with a reddish tinge. These

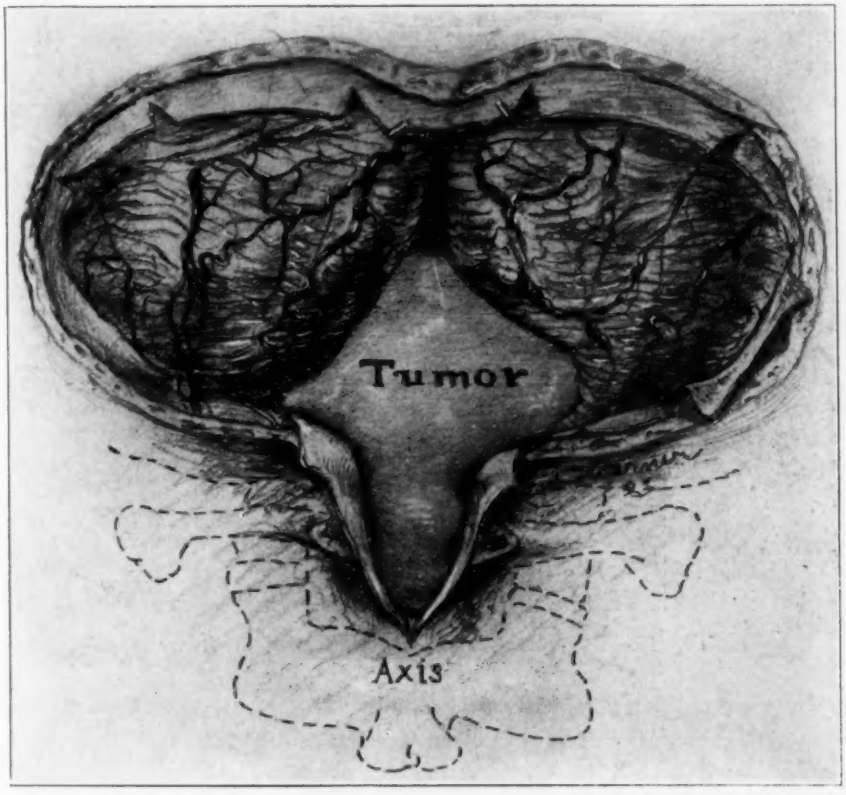

Fig. 9.—Drawing of the appearance at operation of the tumor in Case 4.

larger granules possibly have some relationship to the first ones, because gradations may be seen between them in the same cell.

In the denser portions fibrils are few, but in the looser areas are large numbers of them, many of which stain poorly with stains for neuroglia fibrils. They are clearly the prolongations of the pear-shaped cells grouped around the vessels. Blood vessels are few in number, with poorly developed walls. Connective tissue stains fail to show any well-developed collagen.

CASE 4.—*Ependymoma of the fourth ventricle. Suboccipital exploration with partial removal. Recovery.*

G. D.,[10] aged 10, a schoolboy, was admitted Jan. 6, 1923, complaining of headache, dizziness, and stiffness of the neck. In July, 1922, he had a sharp frontal headache, which was increased by hyperextension of the head. About a week later he vomited his breakfast suddenly and without warning. The vomiting continued almost daily until his admission. In December, 1922, he complained of weakness of the arms and soon began staggering, with a tendency to fall forward. Three weeks before admission he complained of diplopia and became so weak he was put to bed.

He was in a sanatorium a month for "constitutional psychopathic inferiority."

Positive Neurologic Findings.—The findings were: Suboccipital tenderness and slight rigidity of the neck; bilateral choked disk, with swelling of 6 to 7 diopters; nystagmus on looking to right and left; marked incoordination of all extremities, of cerebellar type; hypotonicity of all extremities; tendency to

Fig. 10.—Photograph of Case 4 at time of discharge.

ankle clonus on the right. The combination of hypotonicity, incoordination and emaciation was so marked that he could neither sit up in bed nor feed himself.

Operation.—Jan. 17, 1923, Dr. Horrax made a suboccipital exploration with partial tumor removal. The approach was bloody but not particularly difficult. Puncture of the lateral ventricle obtained large amounts of fluid. On opening the dura, a reddish gray tumor was seen between the hemispheres of the cerebellum, but not attached to them, and extending down into the spinal canal (Fig. 9). The arch of the atlas was removed and the dura split down to the

10. This boy was shown at the meeting of the American Neurological Association, May 31, 1923. At that time he could stand on either foot, run up or down stairs or whirl around quickly without any trace of disequilibration, but he still showed hypotonicity of all extremities. He had marked secondary optic atrophy with no elevation of the disks. He had been perfectly well except for occasionally vomiting his breakfast.

axis. The tumor extended down beyond this point. As it was obviously firmly attached to the posterior surface of the medulla, no attempt was made to enucleate the growth. The capsule was split and part of the tissue removed with a pituitary spoon. This caused marked respiratory embarrassment. The wound was then closed.

Postoperative Course.—The patient improved rapidly. By February 10 he could walk around the ward unaided and could read fine print. (On admission he could see only headlines.) The choked disk subsided completely. Headaches and vomiting were completely relieved. He was given intensive roentgen-ray treatments and on February 17 was discharged, walking out unaided although somewhat unsteadily (Fig. 10).

Microscopic Examination.—The general structure of the tumor is well shown in Figure 11. It is made up for the most part of polygonal cells with well defined cell boundaries, at places closely crowded against each other, at others separated by coarse fibrils. In some areas the cells tend to group themselves around the blood vessels. In others they form rings around a minute central canal. For the most part they form a fairly solid mass without definite architecture. Blood vessels are rather few, and connective tissue is confined to their walls. The walls of most of the capillaries show hyaline change, staining very faintly with aniline blue and acid fuchsin.

The coarse fibrils which are present in all parts are quite definitely not neuroglia fibers of the ordinary type, but the prolongations of the cell cytoplasm. They are coarser than neuroglia fibrils and stain for the most part faintly with neutral ethyl violet-orange G and acid fuchsin. (Weigert noted long ago that the ependymal prolongations did not stain by his method.) They course around among the cells and where the cells are grouped around the vessels they run toward the vessel wall to end in a foot on the connective tissue.

Where the cells are grouped in mass, they are polygonal in shape. Around the vessels they are pear shaped, due to their nuclei lying at different levels, with the fibril coming off the central end. They have abundant, very heavy, granular cytoplasm, which in phosphotungstic acid preparations stains almost black, with acid fuchsin-methyl green a light pinkish green, and with neutral ethyl violet-orange G a bright clear orange.

These last preparations are by far the most instructive, for one sees clearly in the cytoplasm the clean-cut markings characteristic of ependymal cells. Some of them are shown in Figure 12, and vary in number from two to a dozen or more in a cell. They vary greatly also in size, and in shape form round granules to bacillus-like rods. Very often a group of them might be mistaken for a nucleus cut tangentially, but this error is easily avoided by staining with acid fuchsin-methyl green when the granules in question are stained bright red and the chromatic material bright green.

In many spots the cells form rings around a small central canal and have a superficial resemblance to the rosettes of "neuroblastomas." The center of the ring in this case, however, is formed by the cell bodies, the coarse fibrils radiating off from the centrifugal ends of the cells. In the ends of the cells bordering on the minute canal at the center of the ring may be seen the markings characteristic of ependymal cells, usually in pairs. No definite cilia were ever seen. The nuclei are large and oval, with abundant chromatin. Two mitotic figures were found in a long search.

Occasionally cells are found with large numbers of much coarser granules which do not stain so sharply with ethyl violet, but have a reddish tinge.

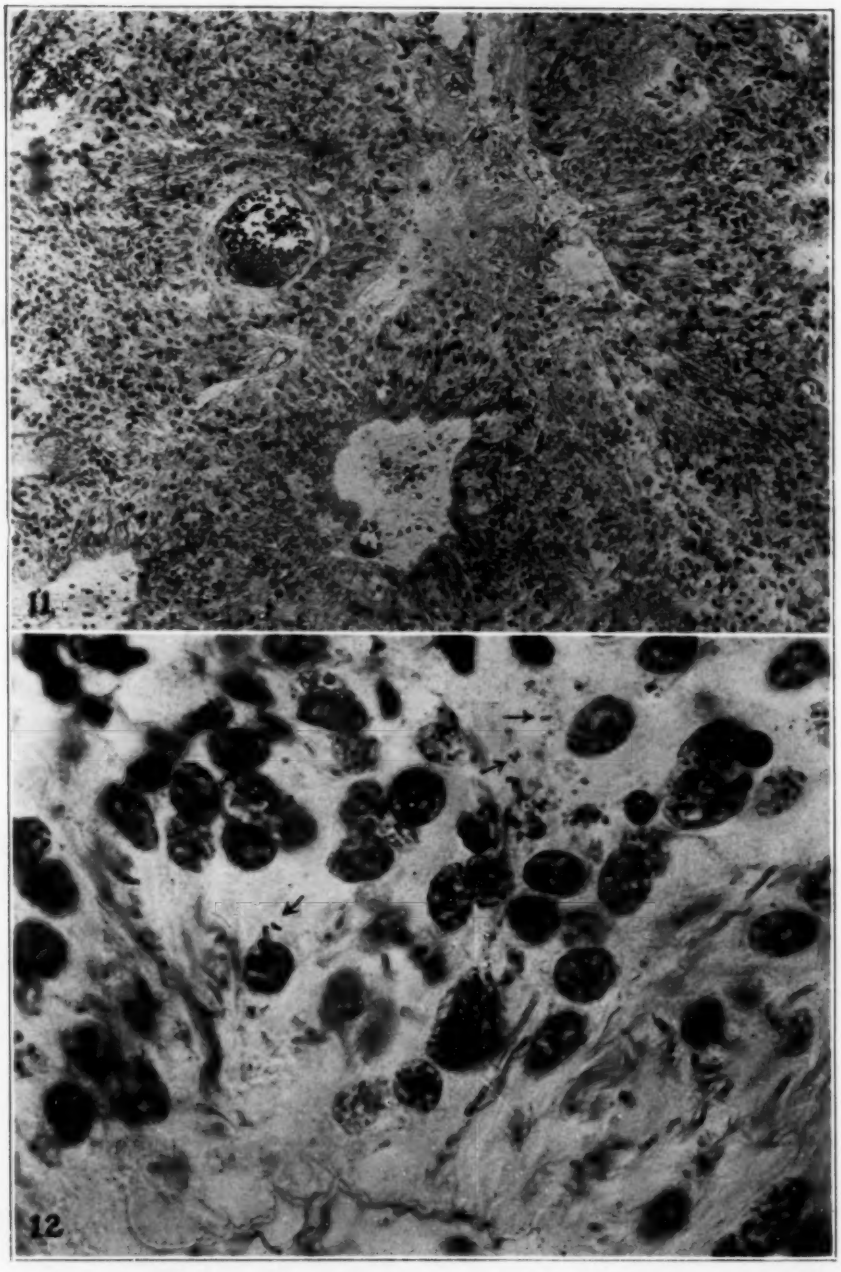

Fig. 11.—Low power photomicrograph showing general structure of tumor in Case 4. Many fibrillae are seen. Neutral ethyl violet-orange G stain; × 100.

Fig. 12.—Higher magnification of tumor in Case 4. Blepharoplasten indicated by arrows. Fibrillae out of focus. Neutral ethyl violet-orange G stain; × 850.

B. EPENDYMOMAS OF THE VELUM MEDULLARE POSTERIUS

The first case was shown at necropsy to be a tumor of the velum medullare posterius. The second must remain questionable in the absence of postmortem verification. Microscopically it would be placed about midway between the ependymomas and tumors of the choroid plexus. Its cells contained typical blepharoplasten; there was practically no connective tissue, and no tendency to papillomatous structure. On the other hand, no neuroglia fibrils were found and the cells were extensively vacuolated. It was not attached to the floor of the ventricle. It seemed to me reasonably certain, therefore, that it had arisen from the velum medullare posterius, whose cells are intermediate in structure between those of the choroid plexus and those of the floor of the fourth ventricle.

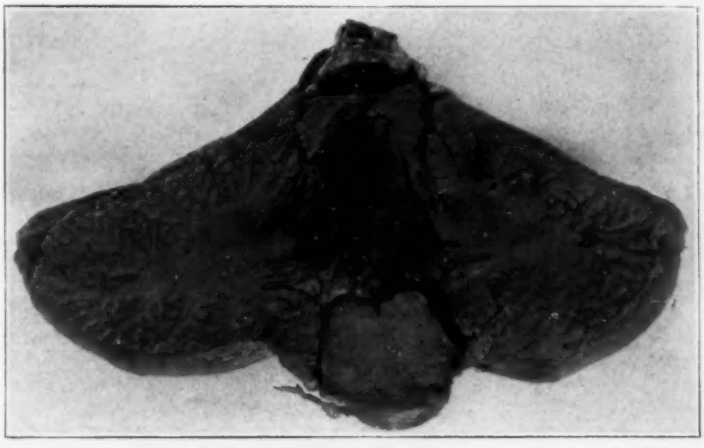

Fig. 13.—Cross section of cerebellum and bulb showing gross appearance of tumor filling fourth ventricle from Case 5.

The similarity of structure of the tumor in Case 5 to that of the tumor described by Muthmann and Sauerbeck is most striking.

CASE 5.—*Ependymoma of fourth ventricle. Unoperated. Death. Necropsy.*

P. S., aged 23, a student, was admitted May 5, 1914, complaining of vomiting, headache, double vision, dizziness and failing vision, having been referred by Dr. J. H. Standard of Hartford, Conn. In July, 1912, he began to have severe headaches and vomiting. The headaches were usually occipital and accompanied by vertigo. He also became very drowsy. In August he complained of diplopia and the visual acuity began to fail. In September, 1912, a right subtemporal decompression was done, which relieved the headache. Acuity of vision had remained stationary but other symptoms had since progressed. In January, 1913, it was noticed that he did not walk very steadily, and slowly he became unable to walk. He was brought to the hospital because lately he had become very weak and lethargic.

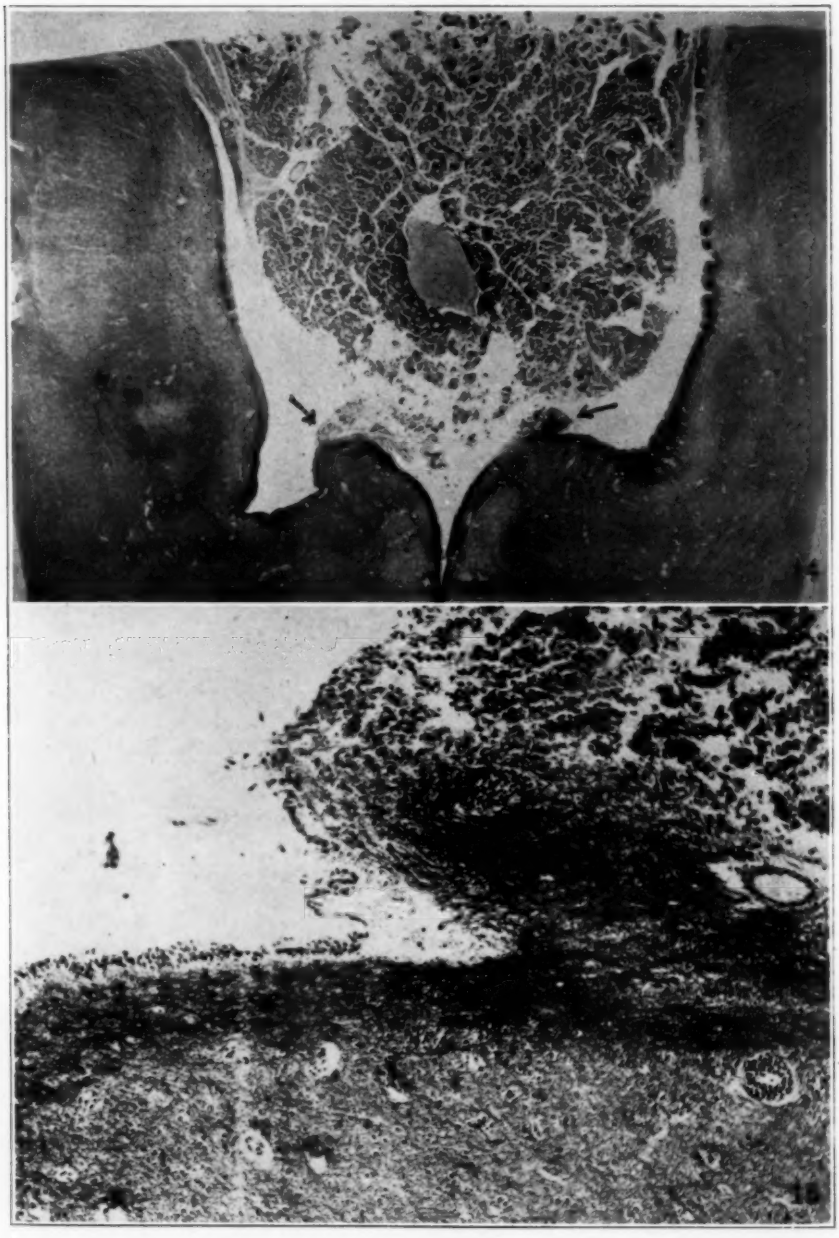

Fig. 14.—Low power photomicrograph of tumor from Case 5, showing points of attachment of tumor to taeniae of the fourth ventricle. Phosphotungstic-acid hematoxylin stain; × 5.5.

Fig. 15.—Low power photomicrograph from same section as Fig. 14, showing transition of ventricular ependyma into tumor at taenia; × 80.

Positive Neurologic Findings.—The decompressed area was bulging about 4 cm. The patient was somewhat drowsy and had bilateral secondary optic atrophy. The right pupil was larger than the left. Both were constricted and did not react to light. There were also paralysis of conjugate movements of the eyes, weakness and spasticity of both lower extremities, bilateral ankle clonus and a dubious Babinski sign on either side.

Clinical Course.—May 15, 1914, ventricular puncture in the parietal region on the right side under local anesthesia demonstrated the presence of rather advanced hydrocephalus. Since interference seemed inadvisable, no further operative procedure was undertaken. The decompressed area remained soft, but the patient became more and more stupid and untidy and died rather suddenly from respiratory failure, July 1, 1914. A necropsy was obtained.

Gross Appearance of Tumor.—The growth completely filled the fourth ventricle and its cut surface had a ragged, granular appearance, rather reddish-brown in color (Fig. 13). It extended through the aqueduct of Sylvius into the third ventricle and was attached at either side to the taeniae of the fourth ventricle (Fig. 14). Above it was continuous with an enlarged pineal gland. The transition of the normal ependyma into the tumor at the taeniae (Fig. 15) leaves no doubt that it had originated from the velum medullare posterius.

Microscopic Examination.—The tumor is a mosaic of polygonal cells with well defined cell boundaries. Thin walled capillaries are rather numerous. Nowhere are there any fibrils, but each of the cells around the capillaries sends down a long, narrow, ribbon-like projection to the vessel wall. These projections do not stain more heavily than the rest of the cytoplasm with stains for neuroglia fibrils. They show very faintly in Figure 16, at the bottom, and form very striking fan-like areas around the vessels.

The cells have large vesicular nuclei, and in practically every one may be found the characteristic markings, usually two, side by side, but sometimes three or more. Occasionally cells will be found heavily loaded with coarser granules staining with a more reddish cast (Fig. 17).

Connective tissue is confined to the thin walls of the capillaries. Neuroglia fibrils are found only where the tumor reaches the brain wall. Here they extend a short way into the growth as a kind of pedicle.

The medulla oblongata shows marked fibrosis, especially of the subependymal layer, which bulges out at intervals, giving the floor of the fourth ventricle a corrugated appearance. The ependyma of the floor is a single layer near the midline, but as the tela chorioidea ventriculi quarti is approached laterally it becomes many layered, to spread out suddenly at the taenia and transform itself into the tumor (Fig. 15).

CASE 6.—*Ependymoma of fourth ventricle. Operation. Recovery.*

B. S., aged 41, a farmer, was admitted Oct. 27, 1915, complaining of headache, vomiting and staggering gait, having been referred by Dr. Syver Vinje of Hillsboro, N. D. The illness began in August, 1914, with vomiting before breakfast, which has recurred practically every day since. In January, 1915, headaches appeared, not very well localized, accompanied at times by a feeling of tension in the back of the neck. In May, 1915, vision commenced to fail, and a month or so later dizzy spells began, with difficulty in walking, which has steadily increased. For the last two months he had been troubled by double vision.

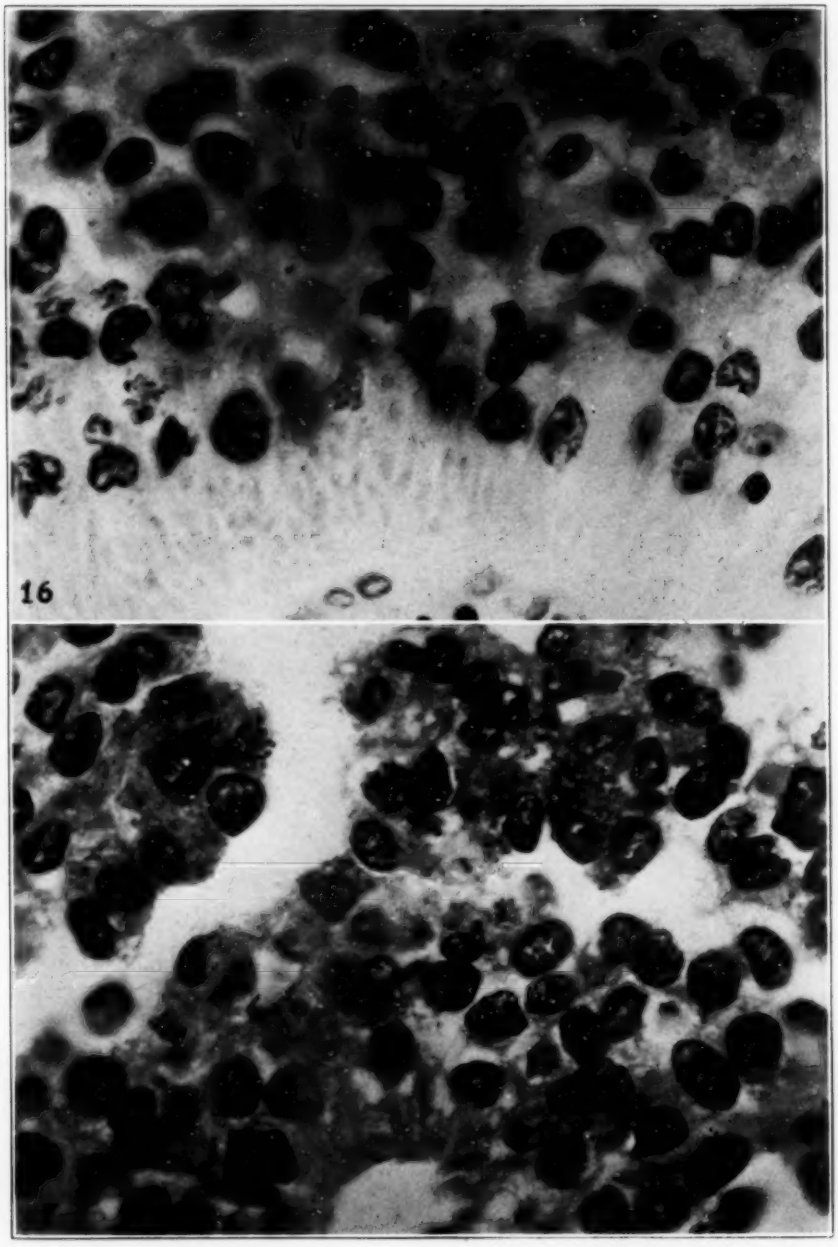

Fig. 16.—Photomicrograph from tumor in Case 5, showing blepharoplasten indicated by arrows. Neutral ethyl violet-orange G stain; × 850.

Fig. 17.—Another field from the same section as Fig. 16 showing cells with coarser granulation.

Positive Neurologic Findings.—The findings were: Station and gait uncertain, with tendency to deviate to the left; incoordination of cerebellar type involving all the extremities, especially the legs; nystagmus on looking to the right and left. A caloric test gave less marked reaction from the left ear. Bilateral choked disk was present, with swelling of from 3 to 4 diopters, with secondary atrophy and marked impairment of visual acuity. The reflexes were hyperactive.

Operation.—Oct. 29, 1915, Dr. Cushing made a suboccipital exploration. The approach was difficult because of the thickness and irregularity of the bone and the large venous channels it contained. Puncture of the lateral ventricle lessened the bleeding, and the operation proceeded satisfactorily. The posterior part of the atlas was removed and the dura was incised down to the axis. The lower extremity of a tumor was thus exposed, which lay between the cerebellar hemispheres on the posterior surface of the cord and bulb. It had a curious granular appearance (Fig. 18) and did not seem to be definitely attached. The lower portion was tilted out and removed. No more extensive removal was attempted because of a large transverse vessel, bleeding from which could not have been controlled without damage to the bulb. The wound was closed as usual.

Postoperative Course.—Convalescence was satisfactory and the patient was discharged Nov. 29, 1915. Headache, vomiting, and double vision had entirely disappeared. The choked disk had subsided. Nystagmus, incoordination and staggering gait were practically unchanged. In June, 1916, when last heard from, his condition was about the same as on discharge.

Microscopic Examination.—The tumor consists of an almost solid mass of cells, with here and there small capillaries. Connective tissue is confined to the poorly developed walls of these vessels, about which the tumor cells show little tendency to group themselves.

The cells have definite borders and are apt to be rounded, so that they do not fit closely together, but leave spaces between. The cytoplasm is heavy and granular, but in most of the cells there are rather large vacuoles. The cells along the blood vessels are pear shaped and arranged so that one will have its big end and its two neighbors their small ends on the vessel wall. In practically every one may be found the characteristic markings, usually in pairs of granules, with a clear halo (Fig. 19). Three or more may be found in a single cell. Often, groups of bacillus-like rods are seen. Cells containing masses of larger globules, such as were seen in the previous cases, are nowhere to be found. There is no sign of fibril formation of any kind.

COMMENT

Concerning the clinical picture in the preceding cases little need be said, since it is outside the realm of the present paper. In each case symptoms of intracranial tension were superadded to the signs of a midcerebellar lesion. One might call attention to the fact that vomiting so frequently preceded signs of tension, sometimes for many months. This fact, as well as the persistence of the vomiting after the relief of the pressure symptoms, is easily explained by the attachment of the tumor to the medulla over the vagus center. Of course the early

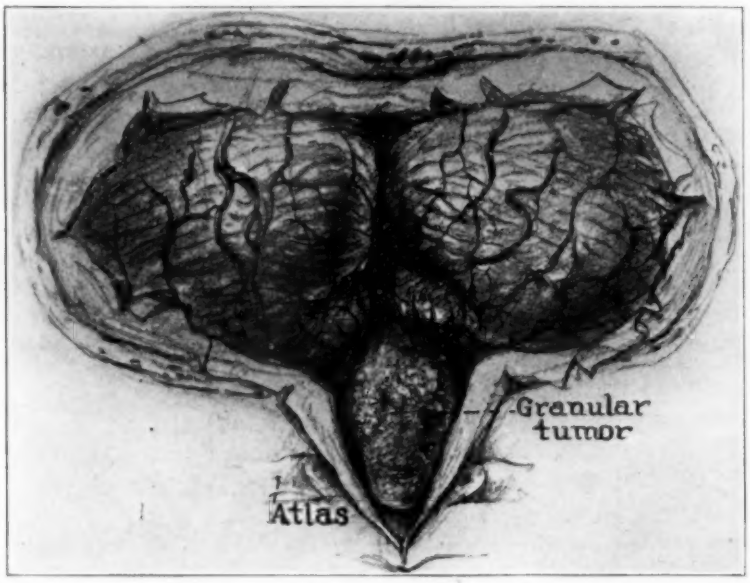

Fig. 18.—Drawing showing gross appearance of tumor from Case 6.

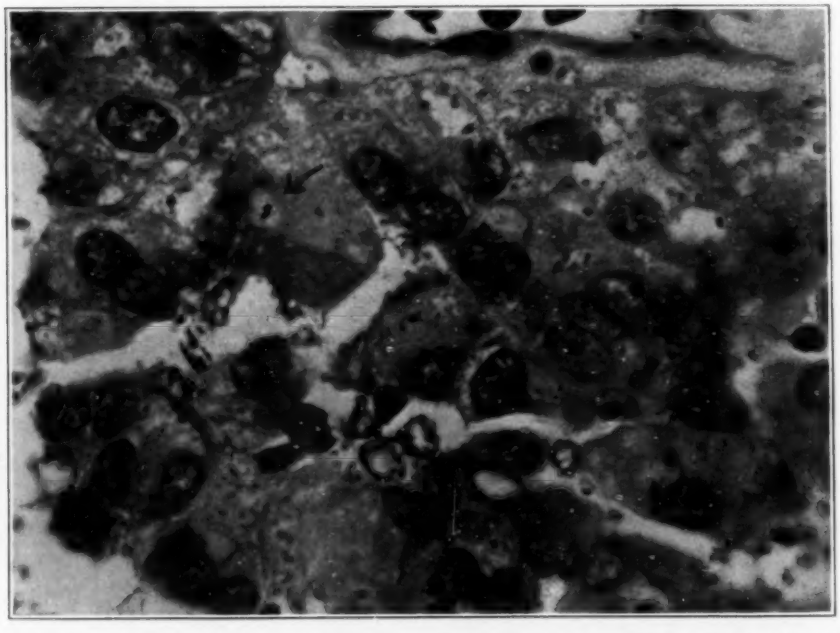

Fig. 19.—Photomicrograph from tumor in Case 6, showing vacuolization of cells and blepharoplasten, indicated by arrows. Neutral ethyl violet-orange G stain; ×850.

development of vomiting cannot be considered pathognomonic of an ependymoma of the fourth ventricle, for it often occurs with other growths in the posterior fossa.

It would seem that these tumors are slow growing and relatively benign. When uncovered at operation they should be left alone. In this series manipulation of the tumor invariably caused respiratory embarrassment and on one occasion fatal hyperthermia. In one instance when the tumor was not disturbed the patient was in excellent health more than ten years later, while another died, almost certainly of acute blockage of the cerbrospinal fluid drainage six months after operation.

In addition to the blepharoplasten, many of the cells of these tumors contained masses of coarser granules, which stained less typically. They are rather banal and often may be found in neuroglia cells. Sometimes pigment granules may be seen. Kraus [11] has investigated extensively these granulations in the case of a small pigmented subependymal glioma from the wall of the lateral ventricle.

The question whether blepharoplasten may be found in other tumors than those arising from ependymal cells must be answered affirmatively. I have seen what appear to me to be blepharoplasten in two other tumors, one from a case of multiple circumscribed tumors of the cortex, and the other a subependymal glioma from the region of the fourth ventricle.

A cross section of the brain from the first case showed three tumor nodules about 1.5 cm. in diameter which were considered to be metastatic carcinoma. Microscopic examination, however, did not show them to be so sharply circumscribed as they appeared to the naked eye, and their cells contained a profusion of minute granules (Fig. 20) having all the characteristics of blepharoplasten, except perhaps their profusion and the absence of a surrounding halo. There were no neuroglia fibrils to be found in these tumor nodules. We must be dealing here with groups of primitive neural tube cells which have attempted to differentiate into ependymal cells and so designate the tumor as a "spongioblastoma ependymale." A similar explanation must also apply to the tumors described by Rosenthal, Stroebe, Marburg and others; in fact, Marburg employs the term "blastoma ependymale."

In the case of a huge subependymal glioma involving the medulla oblongata, cerebellum and inferior cerebellar peduncles, there were found near the ventricular border certain cells containing granules resembling blepharoplasten, although not surrounded by a clear zone in the cytoplasm. As will be seen from the photomicrograph (Fig. 21) these cells must be the same as those found by del Río-Hortega near the ventricles in cases of cerebral softening (compare Figure 2), and

11. Kraus: Virchows Archiv. **217**:121, 1914.

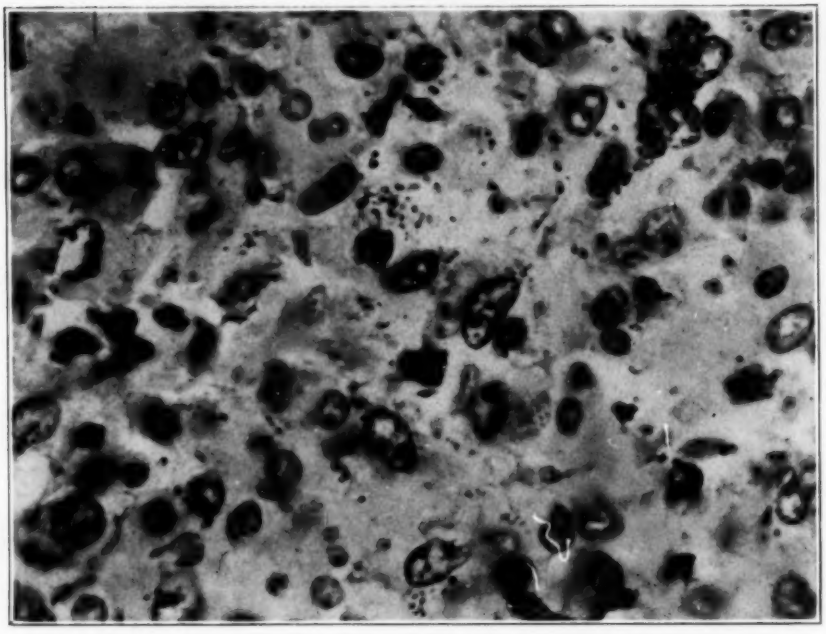

Fig. 20.—Photomicrograph from tumor nodule of a case of multiple tumors of cortex, showing profusion of blepharoplasten. Neutral ethyl violet-orange G stain; × 850.

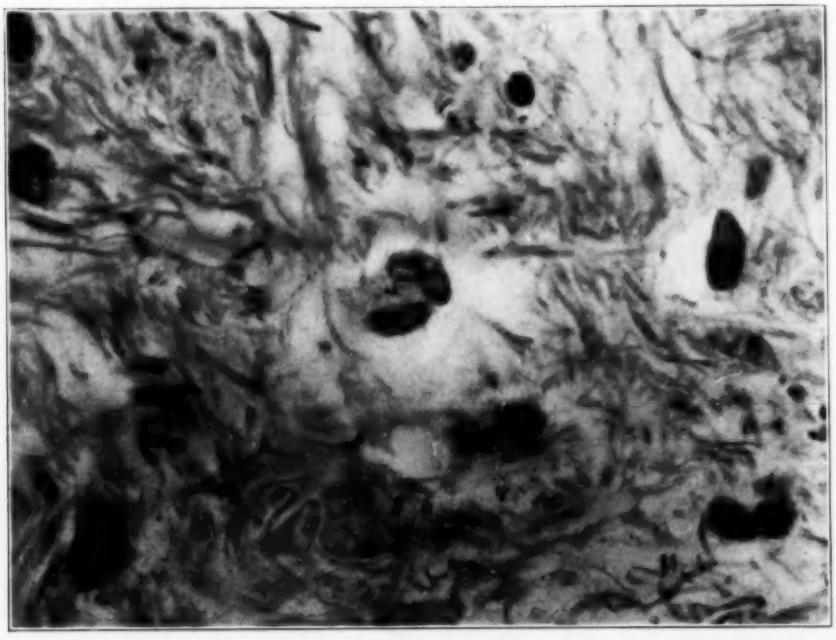

Fig. 21.—Cell from ventricular border of a subependymal glioma. Granules resembling blepharoplasten are seen. Neutral ethyl violet-orange G stain; × 850.

undoubtedly arose from the ependymal cells. That the tumor itself is composed of subependymal glia is evident.

SUMMARY

It seems, therefore, that the microscopic structure typical of an ependymoma may be summarized as follows:

The tumor is composed of a mosaic of polygonal cells with well defined cell borders and heavy, coarsely granular cytoplasm. The cells are crowded against each other and have a single, large, vesicular nucleus. Among them may be seen a few coarse fibrils which stain poorly with methods for the demonstration of neuroglia fibrils. In certain areas the fibrils may be seen to be the continuation of one extremity of the cells. In these areas the cells are apt to have a tendency to arrange themselves radially around blood vessels or around channels empty except for a protein coagulum. The blood vessels are broad channels whose walls contain a little poorly developed, often hyalinized connective tissue. No other connective tissue is present. In practically all the cells (probably all if the entire cell were included in the section) is to be seen a clear area containing sometimes one, usually two or three, rarely as many as a dozen small, clean-cut markings which stain heavily by methods for demonstrating neuroglia fibrils, and may be spherical or short bacillus-like rods. These markings we have interpreted as blepharoplasten, so typical of ependymal cells.

DISCUSSION

Dr. William G. Spiller, Philadelphia: I am impressed by the large number of ependymomas that Dr. Bailey has reported. I have always regarded the ependymoma as one of the rarest tumors. I should like to ask Dr. Bailey during what period of time these six cases came under observation. In 1907 I reported two cases of ependymoma. As Dr. Bailey has stated, one could see in the tumor of the fourth ventricle distinct processes growing out from the brain tissue surrounding that portion of the ventricle, and these processes were covered with ependymal cells. I do not know any other case similar to it, because it gave ependymomatosis throughout a large part of the pia of the spinal cord. Since then I have studied another case of ependymoma, which had been in the service of Dr. Mills; the tumor was in the cervical portion of the spinal cord, and had no connection with the fourth ventricle.

Dr. Bailey, in closing: These are all I could find in Dr. Cushing's collection of verified brain tumors, consisting of practically nine hundred cases, so that they occur perhaps once in a hundred cases of brain tumor.

A NOTE ON THE COMPARATIVE HISTOPATHOLOGY OF ACUTE ANTERIOR POLIOMYELITIS AND EPIDEMIC ENCEPHALITIS *

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University of Illinois
CHICAGO

Epidemic encephalitis and epidemic poliomyelitis are typical instances of acute inflammation of the central nervous system. In both conditions the entire nervous system is more or less involved, the mesencephalon principally in the former, and the spinal cord in the latter. The site of the lesion is thus the main aid in the histopathologic diagnosis of typical cases. In atypical cases, both the mesencephalon and the spinal cord may be involved, and such cases present great difficulties in differential diagnosis. In fact the similarity in localization, gross character and histology may be so great that, as Häuptli [1] pointed out, they might be considered as identical processes.

However, it has been shown by clinical, serologic and possibly also bacteriologic studies that they are distinct morbid entities.

My studies of a case of acute anterior poliomyelitis (Landry's type) and of one of epidemic (lethargic) encephalitis have led me to the conclusion that these two diseases also differ from each other histologically; that in spite of the close similarity in the localization and the gross character of the lesion each possesses more or less specific features.

REPORT OF CASES

CASE 1.—The case of epidemic encephalitis has been recorded in a previous contribution.[2] It concerned a female infant aged 4 weeks, who vomited repeatedly for two days when recurrent attacks of contraction of the diaphragm set in. When admitted to the Presbyterian Hospital, March 14, 1919, the child was stuporous, with a temperature ranging from 98 to 102 F. and a leukocyte count of 9,950. Laparotomy was performed and an appendicostomy was made. The following day the temperature rose to 103.2 F. and subse-

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* From the Division of Neurology of the College of Medicine, University of Illinois, and the pathologic laboratories of the Illinois State Psychopathic Institute and Cook County Hospital.

* Presented before the Forty-Ninth Annual Meeting of The American Neurological Association, Boston, June 1, 1923.

1. Häuptli, O.: Zur Histologie der Poliomyelitis anterior acuta und der Encephalitis epidemica (lethargica), Deutsch. Ztschr. f. Nervenheilk. **71**:1, 1921.

2. Bassoe, Peter, and Hassin, G. B.: A Contribution to the Histopathology of Epidemic ("Lethargic") Encephalitis, Arch. Neurol. & Psychiat. **2**:24 (July) 1919.

quently ranged between 100 and 102 degrees. A few days later nystagmus and strabismus were observed. The child died on March 25. Microscopic examination revealed a typical encephalitis with involvement of the entire spinal cord. Much as it resembled the cerebropontile type of poliomyelitis, it possessed, as will be shown, somewhat different features.

Case 2.—The case of poliomyelitis was in a boy, aged 12, who was admitted to the Cook County Hospital, in 1922, where he died the following day. The entire duration of the illness was about five days. It began with backache, cold sensation in the hands and great weakness. When brought to the hospital he was cyanotic and sweating profusely. He lay motionless, could not articulate and did not control the bladder and rectum. Consciousness and sensibility were preserved.

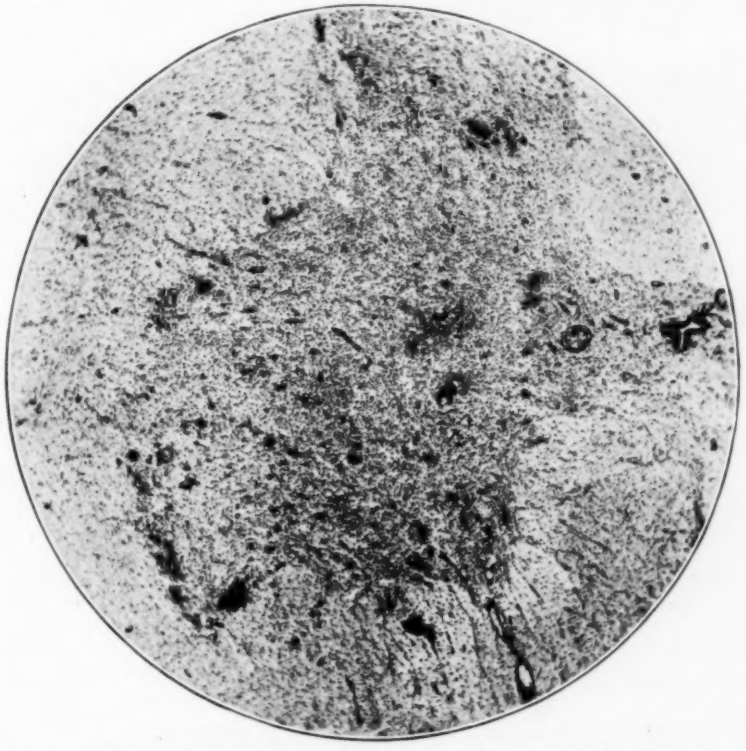

Fig. 1.—Poliomyelitis. Anterior horn region of cord. Intense perivascular exudate and parenchymatous infiltration by glia cells; chiefly in the anterior horn and base of the posterior horn. *C*, central canal. Toluidin blue × 36.

Examination.—The neck, and the upper and lower extremities were completely paralyzed with all reflexes, deep and superficial, lost. The temperature was 102, pulse 92, respiration 24. The pupils reacted normally to light and in accommodation; the spinal fluid was clear, under low pressure and contained 140 lymphocytes, with a moderate amount of globulin. The blood examination revealed 14.400 leukocytes.

Microscopic Examination.—Studies of the spinal cord and brain hardened in formaldehyd solution revealed: (1) marked cellular infiltration of the pia of

the spinal cord and of the base and convexity of the brain; (2) perivascular infiltration of the gray and white substances of the spinal cord, medulla, pons, midbrain, basal ganglions and the nucleus dentatus of the cerebellum; (3) diffuse parenchymatous changes, with degeneration of the ganglion cells, especially of the spinal cord, and enormous glial proliferation with formation of foci; (4) pure parenchymatous brain changes, and (5) infiltrative changes in the choroid plexus. In general, the findings were quite typical of those repeatedly described as classical of this disease. Only the most striking features will be outlined.

Perivascular and Pial Infiltrations: These consisted mainly of lymphocytes, large mononuclears or lymphoid cells of Wallgren, and of the polyblasts of Maximow. Plasma cells and polymorphonuclear leukocytes were absent. An interesting feature was an infiltration of the smallest capillaries, some of which

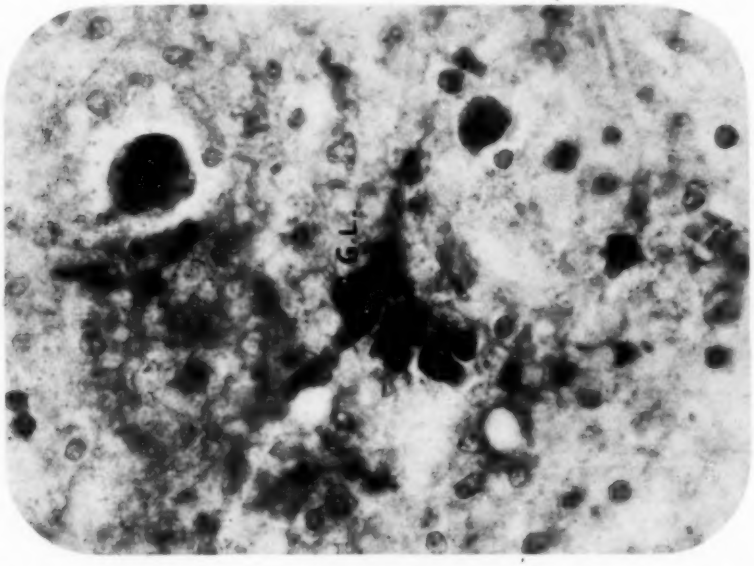

Fig. 2.—Poliomyelitis. Cytoplasmic glia cells in the center. The cell marked *Gl* shows distinct processes. Alzheimer-Mann × 625.

were not larger than a red blood corpuscle. The perivascular infiltrations involved both the gray and the white substances and were equally marked throughout the entire length of the spinal cord. Some blood vessels and capillaries, however, were not infiltrated, but exhibited greatly hypertrophied endothelial cells, well stained, and with nuclei rich in chromatin. Nowhere did the infiltration directly transgress the adventitial spaces of Virchow-Robin.

Parenchyma: The infiltrations of the perivascular spaces were however associated with an infiltration of the parenchyma itself (Fig. 1). This was densely packed with a mass of cell bodies, the nuclei of which were exceedingly polymorphous. In shape the nuclei were round, kidney, horse-shoe or rod shaped with minute droplets of chromatin. The cell bodies often possessed a generous amount of cytoplasm. These cells were either scattered singly or formed dense foci. The foci were especially numerous in the anterior horns, less so in the posterior horns where they occupied the base only.

The character of the foregoing cells has been variously interpreted. Goldscheider [3] considered them "fixed elements," probably "adventitial cells," possibly also "lymphoid or neuroglia" cells; Strauss [4] classified them as endothelial cells, lymphocytes, polymorphonuclear leukocytes and young glia cells; Wickman [5] thought them polyblasts, lymphocytes and glia nuclei; Wallgren [6] considered them glia cells, polyblasts and lymphocytes; and Häuptli interpreted them as glia cells, polymorphonuclear leukocytes and plasma cells.

In the present case the cells are considered to be glia elements exclusively. Some (Fig. 2) exhibited a generous amount of cytoplasm with ramifying processes, that exactly resembled astrocytes. Others did not show ramifications, but only an abundance of cytoplasm; other cell bodies were represented either by small dark pyknotic, or larger nuclei, less densely stained, yet containing

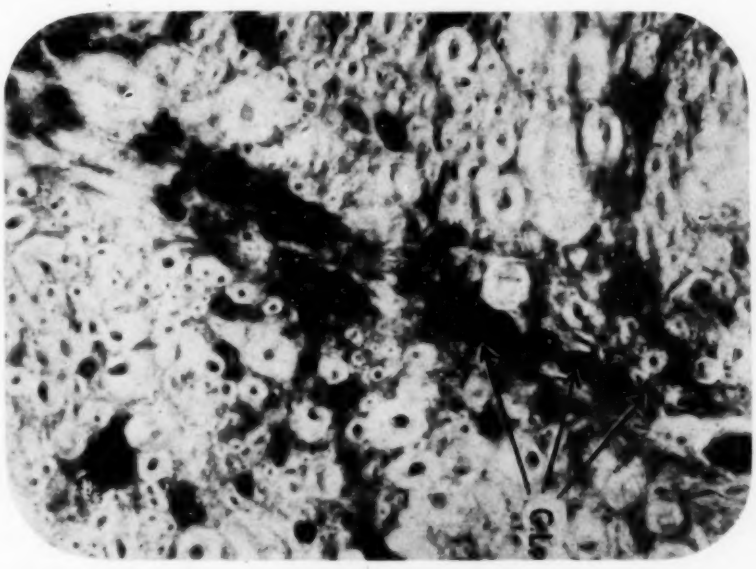

Fig. 3. Poliomyelitis. Anterior root region of cord. Diagonal row of black masses *Gl* are glia cell bodies marking the course of the destroyed anterior root fibers. Alzheimer-Mann × 625.

abundant chromatin granules. All these cell varieties were anchored within the markedly reticulated glia tissue and were in no way connected with the

3. Goldscheider, A.: Ueber Poliomyelitis, Ztschr. f. klin. Med. **23**:494, 1893.

4. Strauss, I.: Epidemic Poliomyelitis. Report of the Collective Investigation Committee on the New York Epidemic of 1907, Monograph 6, Journal of Nervous and Mental Disease Publishing Co., New York: 1910.

5. Wickman, J.: Weitere Studien in Poliomyelitis acuta. Ein Beitrag zur Kenntniss der Neuronophagen und Körnchenzellen, Deutsch. Ztschr. f. Nervenheilk. **38**:396, 1910.

6. Wallgren, Axel: Zur Kenntniss der feineren Histologie und Pathogenese der Heine-Medinschen Krankheit. Arb. aus dem pathologischen Institut der Universität Helsingsfors, New series **1**:81, 1913.

blood vessels or capillaries. In the polymorphism of their nuclei, they very much resemble cell forms, unquestionably glial in origin, that occur in so-called diffuse cerebral sclerosis and have been so well pictured by Uyematsu[7] among others.

The glial nature of these cells could be determined best when studied in regions that were but mildly infiltrated, as, for instance, in the posterior horns, corpora quadrigemina and nucleus caudatus. In the latter, particularly in the subependymal region, they were more or less uniformly distributed and wholly independent of the vessels. They could also be followed in the white

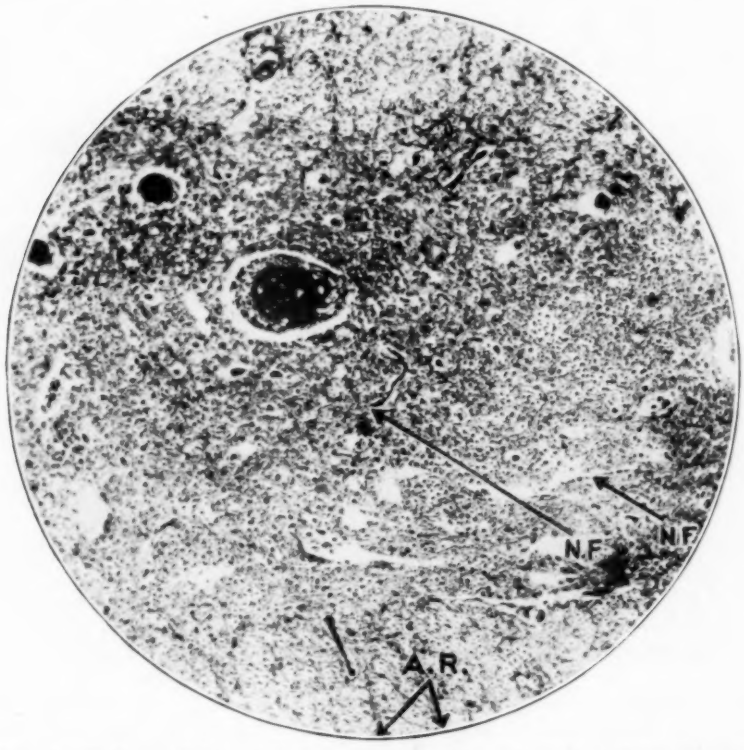

Fig. 4.—Poliomyelitis. Anterior horn region of cord. Note absence of nerve cells and meshwork of fibers; compare with Figure 7. *A.R.*, anterior root fibers; *N.F.*, neurofibrils. Bielschowsky × 110.

substance of the spinal cord especially in the course of the anterior root fibers (Fig. 3) and their glial septa.

Such changes were present throughout the entire white substance of the cord, though they were somewhat less marked in the posterior columns. Regressive changes, such as ameboid glia, foci of softening and hemorrhagic areas were nowhere found.

Ganglion Cells: These showed exquisitely degenerative or regressive phenomena and were strikingly few in number. Many sections showed prac-

7. Uyematsu, S.: A Case of Diffuse Cerebrospinal Sclerosis, J. Nerv. & Ment. Dis. **51**:514, 1920.

tically no ganglion cells, the few present in some sections having been profoundly changed. Some were markedly swollen with the nucleus pale and dislocated; some were liquefied and without traces of Nissl bodies; the fibrils appeared agglutinated and displaced to the periphery, but distinct neuronophagic phenomena were rather rare. When present, the neurophages were glia cells, which formed the dense foci, already described, that marked the former location of ganglion cells. In the posterior horns and the columns of Clarke the ganglion cells were better preserved though somewhat changed, exhibiting cloudy swelling and central chromatolysis.

Fig. 5.—Poliomyelitis. Central canal of cord. The lumen shows granular material and numerous hematogenous elements among which polyblasts with pale curved nuclei predominate. Reproduced with higher magnification in Figure 6.

Axis Cylinders: The delicate nerve fibers that are so abundant in the gray matter of normal spinal cords appeared equally degenerated. Their condition was studied by the methods of Weigert-Pal, Alzheimer-Mann, Bielschowsky, and by the last two methods combined. Like the ganglion cells the axis cylinders were strikingly few in number in the gray matter. Many sections showed but a few axons, which were thinned, tortuous, broken up into minute fragments and for the most part deprived of myelin (Fig. 4). Equally sparse were the nerve fibers in the anterior roots, in which a few single fibers could occasionally be seen (Fig. 4 *A R*). The course of the roots was often marked by a number of hypertrophied glia cells (Fig. 3). The axons and

medullary sheaths in the white columns studied in transverse and longitudinal sections, stained by various methods, showed no noteworthy changes.

The parenchymatous changes, confined to the gray matter especially of the ventral horns, thus involved the entire motor neuron. It is truly remarkable that within so short a space of time—about five days—a destruction of the entire neuron had occurred and had been followed, as is the general law, by proliferative glial changes.

Central Canal: This showed quite interesting changes. It appeared patent (Figs. 5 and 6) and contained an amorphous mass and a number of cell bodies (lymphocytes, polyblasts and so-called lymphoid cells of Wallgren). They resembled exactly the elements within the infiltrated perivascular spaces. The

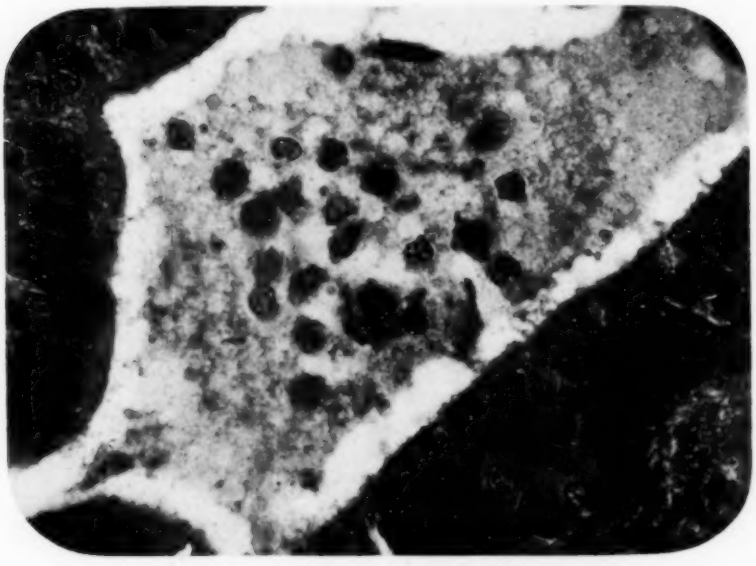

Fig. 6.—The same section as Figure 5 × 625.

ependymal cells were well developed, rich in chromatin, cytoplasm and processes that formed a wall surrounded by numerous larger and smaller vessels, all densely infiltrated.

Medulla, Pons, Midbrain and Basal Ganglions: The changes outlined (ectodermal and mesodermal) were likewise observed in the medulla, pons, midbrain, basal ganglions and to some extent in the cortex. The perivascular infiltrations were enormous (Fig. 7), the infiltrating cells being, as in the spinal cord, lymphocytes and polyblasts, but not polymorphonuclears nor plasma cells.

As in the spinal cord, the smallest capillaries were infiltrated and the parenchyma was densely covered with polymorphonuclear glia cells and numerous glia cell foci. The pigmented cells of the substantia nigra were for the most part preserved; some showed slight changes—cloudy swelling, chromatolysis, and mild neuronophagia. The same infiltration of the smallest capillaries with parenchymatous changes—regressive in the ganglion cells and

progressive in the glia—were present in the medulla, corpora quadrigemina, basal ganglions and nucleus dentatus cerebelli. The entire subependymal area of the caudate nucleus was covered with a mass of cell bodies, more or less uniformly distributed, while the perivascular infiltration was insignificant or absent altogether.

Cortex and Cerebellum: Changes in the ganglion cells were quite mild, though many cells appeared vacuolated, with chromatolysis, neuronophagia and cloudy swelling. Perivascular infiltration was confined to the larger vessels, while the smaller vessels and capillaries exhibited hypertrophied endothelial

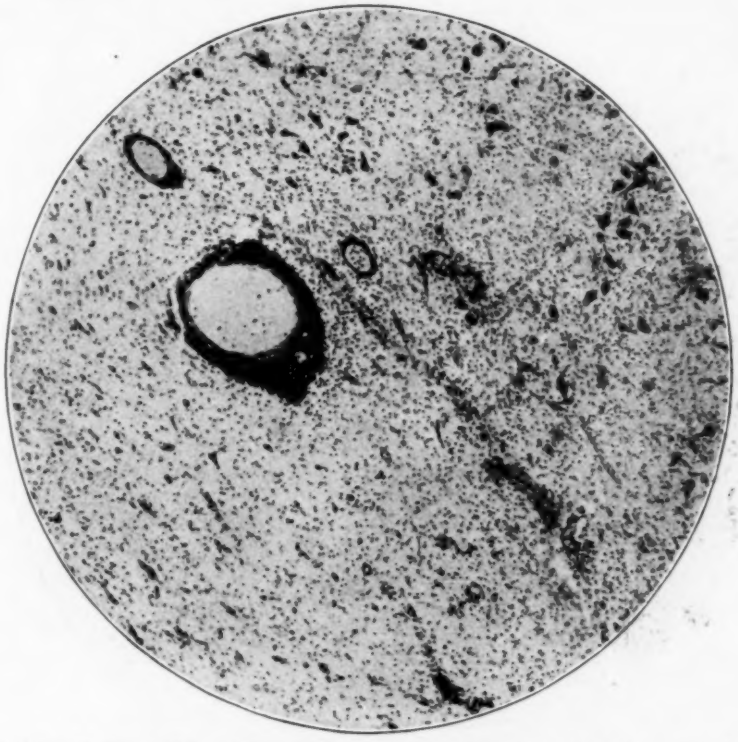

Fig. 7.—Poliomyelitis. The region of the locus coeruleus is covered with densely infiltrated blood vessels. Toluidin blue × 60.

cells that were densely stained. In accordance with the mild ganglionic changes, the glial proliferation was also mild; the nuclei were not proliferated, nor polymorphous as in the spinal cord, the glia tissue generally appearing reticulated.

Pia and Choroid Plexus: The pia showed changes throughout the spinal cord and brain. In the former it was markedly infiltrated, especially in the anterior fissure, with lymphocytes and polyblasts. Around the base, the pons and cerebellum, there were, in addition, numerous mesothelial cells and a few polymorphonuclear leukocytes, while around the occipital lobe there were also fibroblasts and many macrophages packed with green pigment and degenerated lymphocytes. These were confined to the neighborhood of hyperemic veins. Pigment globules were also present in the mesothelial cells which were

greatly hypertrophied. Some of these contained only small droplets of pigment, some larger, while some were packed with them. Other mesothelial cells, away from the blood vessels, not only were hypertrophied, but formed more or less dense foci. Around the cornu ammonis and the motor area the pia-arachnoid appeared very densely infiltrated, the infiltrating cells being not only lymphocytes, polyblasts and mesothelial cells, but also plasma cells. In general, the majority of infiltrating cells of the pia-arachnoid were mesothelial cells; lymphocytes and polyblasts were fewer in number. The stroma appeared greatly infiltrated with fibroblasts (Fig. 8), many of which contained minute droplets of pigment. The veins were distended and hyperemic; many were surrounded by dense foci of fibroblasts, and some were calcified. The tuft cells

Fig. 8.—Poliomyelitis. The choroid plexus. The black foci are masses of fibroblasts around blood vessels.

were swollen, reticular and contained droplets of pigment. In addition, there were foci of hemorrhage and numerous corpora arenacea.

SUMMARY OF FINDINGS

Changes in cells of both ectodermal and mesodermal origin were present throughout the entire central nervous system. The former were represented by degenerative phenomena in the ganglion cells of the spinal cord, medulla and midbrain, and in the nerve fibers of the ventral horns and anterior roots, and by marked proliferative phenomena in the

glia. The latter were represented by perivascular infiltration of the gray and white substances of the spinal cord, midbrain, basal ganglions and to some extent in the brain and the cerebellum. In addition there were widespread reactive phenomena in the pia-arachnoid and the choroid plexus.

THE CONTRAST BETWEEN THE CHANGES IN THE CASES OF POLIOMYELITIS AND EPIDEMIC ENCEPHALITIS

In the case of epidemic encephalitis the changes evident were similar in kind to those in poliomyelitis. The spinal cord, for instance

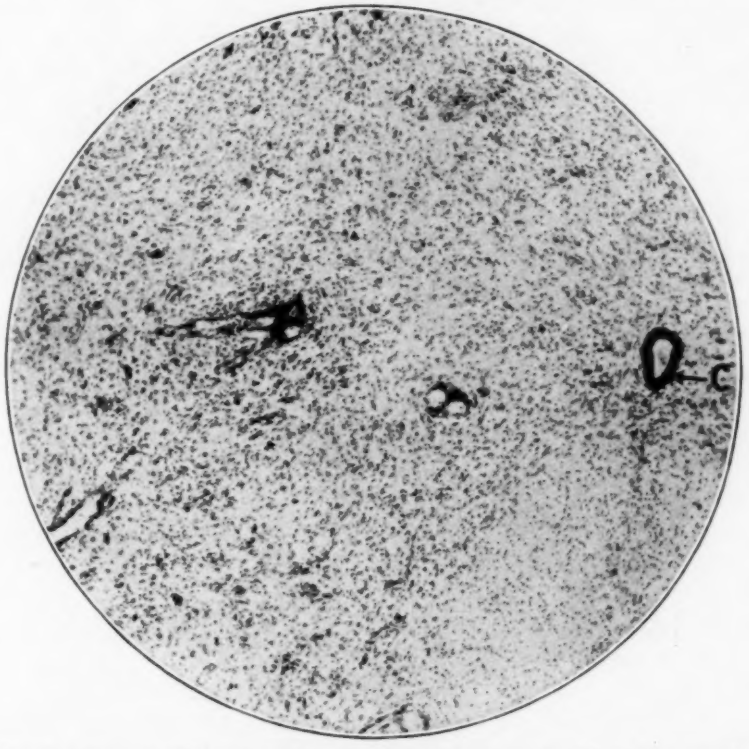

Fig. 9.—Encephalitis. Anterior horn region of cord. Moderate exudate in large vessels and moderate diffuse infiltration of parenchyma. Compare with Figure 1. *C*, central canal. Toluidin blue × 60.

(Fig. 9), contained greatly infiltrated blood vessels in both gray and white substances. The pia, especially in the anterior fissure, was enormously infiltrated, the arteries and veins being involved alike. The infiltrating cells were lymphocytes, polyblasts and quite frequently plasma cells. But neither the extent nor the intensity of the infiltration was so great as in poliomyelitis, and there was a remarkable lack of infiltration of the capillaries. On the other hand, there were distinct

signs of vascular proliferation in the form of an abundance of young capillaries and capillary buds. The sections therefore, appeared much more vascular than those of the poliomyelitis case.

The parenchymatous infiltration in the encephalitis case, which was less marked than in the poliomyelitis case, consisted of glia cells; the anterior horns showed fewer cell bodies and appeared less invaded. Foci of glia cells, though present, were much less numerous. Another noteworthy feature was that both anterior and posterior horns were

Fig. 10.—Encephalitis. Anterior horn region of cord. Note nerve cells and wealth of fibers. Compare with Figure 4. *A.R.*, anterior root fibers. Bielschowsky × 120.

equally involved in contrast to the poliomyelitis case, in which the anterior horns were principally affected.

The mild glial proliferative phenomena were associated with equally mild changes in the ganglion cells. The majority of these cells were well preserved (Fig. 10), though some exhibited changes, such as cloudy swelling, mild chromatolysis and occasional neuronophagia. These changes, again, were observed in both the anterior and the posterior horns. The network of delicate nerve fibers, destroyed in the

poliomyelitis case, were here well preserved, as were the anterior root fibers (Fig. 10).

The proliferative glial phenomena in the white columns in the encephalitis case were insignificant.

The medulla, pons and basal ganglions exhibited changes similar to those in the poliomyelitis case except that the capillary infiltration was lacking. Glia cell foci were present especially in regions where marked ganglion cell destruction had occurred. For instance, in the region of the aqueduct of Sylvius, the cell destruction was very pronounced and there was an enormous glia cell proliferation with formation of numerous foci. The same may be said of the nucleus caudatus in which the cell destruction was much more extensive than in the poliomyelitis case and the subsequent glia cell proliferation was more in evidence.

The central canal contained, in both cases, lymphocytes and polyblasts.

The cortex and the cerebellum exhibited mild parenchymatous and vascular changes, the blood vessels being less abundant than in the case of poliomyelitis.

The pia-arachnoid—somewhat similarly affected in both—showed numerous lymphocytes and mesothelial cells.

The choroid plexus showed mild infiltration of the stroma.

COMMENT

The findings in the two cases differ from those recorded in the literature. That the brain stem may be involved in poliomyelitis is well known.[8] Yet, I know of no case that showed such a wealth of changes in the central nervous system outside the spinal cord as the one here recorded. The reverse is true of the epidemic encephalitis case. Spinal cord changes in the latter have been recorded by von Economo,[9] Harbitz,[10] Stern,[11] Häuptli,[1] Tabler,[12] Winkelman and Weisenburg,[13]

8. Proschkin, G.: Untersuchung eines Falles von sporadischer akuter Poliomyelitis, Ziegler's Beitr. z. Pathol. Anat. **53**:38, 1912.

9. Economo, C.: Encephalitis lethargica, Wien. klin. Wchnschr. **30**:581, 1917; Neurol. Centralbl. **36**:867, 1917.

10. Harbitz, F.: Ueber die Encephalitis lethargica, Ziegler's Beitr. z. pathol. Anat. **67**:458, 1920.

11. Stern, F.: Die Pathologie der sogenannten Encephalitis lethargica, Arch. f. Psychiat. **61**:621, 1920; Die epidemische Encephalitis, Berlin: 1922.

12. Tabler, Th.: Pathologische Beiträge zur Kenntniss der akuten, herdförmig disseminierten, nichteitrigen, vorwiegend lymphocytären, infektiös—toxischen, epidemischen Polioencephalomyelitis (Encephalitis lethargica), Schweiz. med. Wchnschr. **50**:446 and 470, 1920.

13. Winkelman, N. W., and Weisenburg, T. H.: A Case of Myeclonic Lethargic Encephalitis with Unusual Pathologic Manifestations, Arch. Neurol. & Psychiat. **5**:224 (Feb.) 1921.

Calhoun,[14] Gerlach,[15] Spiller,[16] Levaditi [17] and Harris.[18] In none of these cases was the spinal cord involved to the extent here recorded, while in some instances the spinal changes were of an entirely different nature. In general, the two cases here reported resemble each other so much histologically that from the appearance, for instance, of the medulla, pons and other regions, a differential diagnosis is not possible.

Differentiation is, however, possible when the spinal cord changes are contrasted. In both there were marked infiltrative phenomena, though much more marked in poliomyelitis, in which the smallest capillaries were affected. However, these may be spared even in severe cases of poliomyelitis. Therefore, the intensity of the mesodermal changes is not sufficient for differential diagnosis. Of far greater significance are the changes in the parenchyma of the cord. This is decidedly more affected in poliomyelitis than in epidemic encephalitis. More ganglion cells are destroyed in the former and within a much shorter period of time (five days in the present case) than in epidemic encephalitis (the duration in the present instance was thirteen days), the destruction involving also the nerve fibers of the anterior horns and their roots. This destruction was followed by an enormous glia cell proliferation. Thus, both inflammatory and degenerative phenomena, confined to the motor neurons, were in evidence in the poliomyelitis case, whereas in the epidemic encephalitis case there was also involvement of sensory neurons.

The difference in the degree of the degenerative process may be due to a difference in the virus. This is evidently more virulent, in poliomyelitis than in epidemic encephalitis. It therefore provokes more marked and more destructive histopathologic changes.

A few words may be said as to the condition of the central canal. In both cases it contained an amorphous substance in addition to a number of cell bodies that much resembled those present in the perivascular spaces of the parenchyma. Their presence in the central

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14. Calhoun, H.: Histopathology of the Brain and Spinal Cord in a Case presenting a Postinfluenzal Lethargic Encephalitis Syndrome, Arch. Neurol. & Psychiat. **3**:1 (Jan.) 1920.

15. Gerlach, W.: Ueber Rückenmarksveränderungen bei Encephalitis lethargica, Berl. klin. Wchnschr. **57**:585, 1920.

16. Spiller, W. G.: Epidemic Encephalitis with Myelitis, Arch. Neurol. & Psychiat. **7**:739 (June) 1922.

17. Levaditi, C.: Poliomyélite, encéphalite, herpès, Paris: 1922.

18. Harris, Ada F.: Report of a Case of Epidemic Encephalitis with Cord Changes Suggestive of the Possibility of an Early Syringomyelia, Am. J. Psychiat. **78**:679 (April) 1922.

canal in poliomyelitis was pointed out by Walter.[19] In one instance he found only a structureless mass filled with dark granules. In another he found eight cells: three lymphocytes, two plasma cells and three large protoplasmic cell bodies with pigmented nuclei. He does not comment on the mode of their entrance into the canal.

As they exactly resembled the cell bodies of the infiltrated perivascular spaces of the blood vessels, many of which surrounded the central canal, it seems proper to assume they entered from these spaces. This would indicate that the perivascular spaces of Virchow-Robin are in communication not only with the subarachnoid space but also with the central canal; that the flow of tissue fluids, or of the contents of the adventitial spaces, is toward both the subarachnoid space and the spinal canal. The fluid discharged into the subarachnoid space tends to provoke reactive phenomena there which probably vary according to the character of the toxins carried by the fluids. In both poliomyelitis and epidemic encephalitis, especially in the former, there was present in the subarachnoid space a marked increase in mesothelial cells and hematogenous elements. This again might be explained by the greater virulence of the poliomyelitic virus. Whatever the reactive phenomena in the pia-arachnoid, the changes are secondary to those in the parenchyma; this is contrary to the assertion of Harbitz [10] that the meninges are primarily involved, the parenchyma only secondarily as the result of spreading of the inflammation from the meninges.

CONCLUSIONS

1. Poliomyelitis and epidemic encephalitis both exhibit inflammatory and degenerative changes.

2. These are much more intense in poliomyelitis, in which, within a very short time, they cause degeneration of the entire motor neuron, including the dense network of delicate white fibrils of the anterior horns and roots.

3. The degeneration is associated with a marked proliferation of glia cells.

4. The inflammatory as well as the degenerative changes may involve, in both diseases, the entire central nervous system, decreasing in severity: cephalad in poliomyelitis, and caudad in encephalitis.

5. Both the extent and the intensity of the changes may be so similar, that a differential diagnosis is possible only from the degree of the degenerative changes in the cord.

6. The presence of hematogenous elements in the central canal may be interpreted as a proof that the flow of the contents of the perivascular spaces is not only toward the subarachnoid space, but also toward the central canal.

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19. Walter, R.: Zur Histopathologie der akuten Poliomyelitis, Deutsch. Ztschr. f. Nervenheilk. **45**:79, 1912.

DISCUSSION

DR. WALTER F. SCHALLER, San Francisco: I should like to ask Dr. Hassin whether he differentiated the two diseases that he describes by the extent and severity of the lesions or by cell differentiation?

DR. HASSIN: I differentiated between the two diseases by the extent of the lesions.

FAMILIAL MUSCULAR DYSTROPHY

STUDIES OF SIX CASES IN ONE FAMILY

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The literature concerning muscular dystrophy abounds in instances of familial involvement. We have found no clinical report of so heavy an incidence in one family as we here report; seven cases of muscular dystrophy (one dead) have occurred among twelve children born to the same parents. Poore [1] reviewed eighty-five cases among which there are thirty-seven instances of two or more in the same family. Among the 220 instances cited by Gowers,[2] 118 occurred in thirty-nine families. Among the cases cited by Erb,[3] 56 per cent. showed familial aspects. In the study of Collins and Climenko,[4] fifty cases are reviewed, twenty of which show hereditary aspects. Eshner [5] has cited twenty cases at the Orthopedic Hospital in Philadelphia, in two instances similar disorders being found in other members of the family. Among the nine cases studied by Janney, Goodhart and Isaacson [6] at the Montefiore Home, there are four instances in which three are affected in the same family, and three instances in which two are affected in the same family.

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1. Poore, C. T.: Pseudo-Hypertrophic Muscular Paralysis, with an Analysis of Cases, New York M. J. **21**:569, 1875.

2. Gowers, W. R.: Pseudo-Hypertrophic Muscular Paralysis; a Clinical Lecture, London: J. & A. Churchill, 1879, p. 66.

3. Erb, W. H.: Dystrophia muscularis progressiva. Leipzig: Vogel, 1891, p. 170. Reprinted from Deutsch. Ztschr. f. Nervenheilk. Vol. 1.

4. Collins, Joseph, and Climenko, Hyman: Clinical Study of Fifty Cases of Muscular Dystrophy. Contributions to the Science of Medicine and Surgery, by the Faculty in Celebration of the Founding of New York Post-Graduate Hospital. Twenty-Fifth Anniversary Number, 1908, p. 360.

5. Eshner, A. A.: Contribution to the Study of Muscular Dystrophies, Am. J. Med. Sc. **116**:307 (Sept.) 1898.

6. Janney, N. W.; Goodhart, S. P., and Isaacson, V. I.: Endocrine Origin of Muscular Dystrophy, Arch. Int. Med. **21**:188 (Feb.) 1918.

Cases in which it is specifically stated that no similar affections were found in other members of the family are rare. Such cases are reported by Beling,[7] Hamill,[8] Naccarati[9] and Friedman.[10]

Some studies emphasize the familial and hereditary aspects of the disorder. Meryon[11] reported two cases in a family of sixteen. Lutz[12] reported six cases in two generations of one family. Eichhorst[13] described thirteen persons affected with muscular dystrophy in six generations in a family by the name of Bessel. Timme[14] reported a family in which fourteen were affected in four generations. Tscherning[15] cited a family of eight, borne by the same mother, but with two husbands, in which four were affected.

The greater incidence of muscular dystrophy in males is an outstanding feature in some studies. Meryon[16] cited a family of four boys and six girls in which the boys were affected but the girls were healthy. Friedreich[17] reported a family, in which four brothers were affected, who died at the ages of 5, 6, 12, and 16, respectively. These boys had two maternal uncles similarly affected who died at 15 and 16, respectively. Gowers[2] reported an instance of a family of ten, three girls and seven boys, in which four of the boys were affected. Poore[1] cited a case with three half-brothers all of whom were afflicted with muscular dystrophy. He also cited a patient, a male, who had

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7. Beling, C. C.: Case of Dystonia Musculorum Progressiva (Oppenheim's New Disease), J. Nerv. & Ment. Dis. **39**:461 (July) 1912.

8. Hamill, P.: Case of Muscular Dystrophy, Proc. Roy. Soc. Med., Sect. Neurol. **9**:81, 1915-1916.

9. Naccarati, Sante: Primary Myopathies, New York M. J. **105**:351 (Feb. 24) 1917.

10. Friedman, E. D.: Case of Progressive Muscular Dystrophy, J. Nerv. & Ment. Dis. **54**:294 (Oct.) 1921.

11. Meryon, Edward: Practical and Pathological Researches on the Various Forms of Paralysis, London: J. & A. Churchill, 1864.

12. Lutz, Christian: Zwei weitere Fälle von sogenannter Muskelhypertrophie. (Lipomatosis luxurians musculorum progressiva), Deutsch. Arch. f. klin. Med. **3**:358, 1867.

13. Eichhorst, Hermann: Ueber Heredität der progressiven Muskelatrophie, Berl. klin. Wchnschr. **10**:497, 1873.

14. Timme, Walter: Progressive Muscular Dystrophy as an Endocrine Disease, Arch. Int. Med. **19**:79 (Jan.) 1917.

15. Tscherning, Rüdiger: Muscular Dystrophy and Dementia Praecox; an Investigation of Their Hereditary Relations, Dementia Praecox Studies **4**:75 (July) 1921.

16. Meryon, Edward: On Granular and Fatty Degeneration of the Voluntary Muscles, Med. Chir. Trans. **35**:73, 1852.

17. Friedreich, Nikolaus: Ueber progressive Muskelatrophie; über wahre und falsche Muskelhypertrophie, Berlin: Hirschwald, 1873, p. 358.

three maternal uncles similarly affected. Bruce [18] reported the disease in two brothers and an uncle. Holmes [19] reported three brothers with a normal sister. With [20] cited a family of three boys and four girls in which the three boys were affected. Shaw [21] reported two cases, brothers, having muscular dystrophy whose sister remained healthy. Funsten [22] reported the family of a woman married twice; by the first marriage there were three boys and one girl; the boys were afflicted with muscular dystrophy. Of the two boys and two girls by her second marriage, the boys alone were afflicted.

Instances are not wanting in which both sexes are affected in the same manner. Poore [1] cited three instances in which two aunts or uncles had been subjects of muscular dystrophy. Funsten [22] cited a family of four, two boys and two girls, all of whom suffered from muscular dystrophy. Greenfield [23] reported a family of thirteen (eight boys and five girls) in which five (four girls and one boy) were afflicted with muscular dystrophy. Gowers [24] cited a family of eleven girls, four of whom suffered from muscular dystrophy; three died at an early age, and four remained healthy.

Many notable instances of other degenerative conditions occurring in the family coincident with muscular dystrophy might be cited. Greenfield's [23] family suffered from congenital cataract as well as muscular dystrophy. Among the thirteen brothers and sisters, two girls were afflicted with muscular dystrophy and congenital cataract, three others (two girls and one boy) suffered from muscular dystrophy alone, and two (one boy and one girl), from congenital cataract alone. Hertz and Johnson [25] cited an instance of progressive muscular atrophy in a patient whose two children suffered from muscular dystrophy. This patient also had a nephew, three years old, showing a tendency to lordosis and frequent falling. Meryon [11] quoted a letter written in 1859

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18. Bruce, A. N.: Muscle Spindles in Pseudo-Hypertrophic Paralysis, Rev. Neurol. & Psychiat. **9**:110, 1911.

19. Holmes, Gordon: On the Spinal Changes in a Case of Muscular Dystrophy, Rev. Neurol. & Psychiat. **6**:137, 1908.

20. With, Otto: Eine Familiäre Atypische Form der Dystrophia Musculorum Progressiva, Freiburg: 1906.

21. Shaw, A. F. B.: Pseudo-Hypertrophic Muscular Dystrophy; Two Cases, Dublin J. Med. Sc. **138**:115, 1914.

22. Funsten, R. J.: Clinical Study of Thirty Cases of Muscular Dystrophy, J. Bone and Joint Surg. **5**:190 (April) 1923.

23. Greenfield, J. G.: Notes on a Family of "Myotonia Atrophica" and Early Cataract, with a Report of an Additional Case of "Myotonia Atrophica," Rev. Neurol. & Psychiat. **9**:169, 1911.

24. Gowers, W. R.: Myopathy and a Distal Form. Diseases of the Nervous System, Lecture IV, Philadelphia: P. Blakiston's Son & Co., 1904.

25. Hertz, A. F., and Johnson, W.: Progressive Muscular Atrophy Associated with Primary Muscular Dystrophy in the Second Generation, Proc. Roy. Soc. Med., Clin. Sect. **6**:177, 1912-1913.

by the father of twelve children, eight boys and four girls. Six boys had died afflicted with a muscular atrophy, and the other two, nine and eight years, respectively, seemed similarly afflicted. One of these boys broke his thigh twice under circumstances which indicated a dystrophy of bone as well as of muscle. The four girls of this family remained unaffected. These cases were not studied clinically.

REPORT OF CASES

This is a study of progressive muscular dystrophy in six persons in a family of twelve children ranging in age from 9 to 32, and of whom eleven are still living. The one who died had been similarly affected for several years.

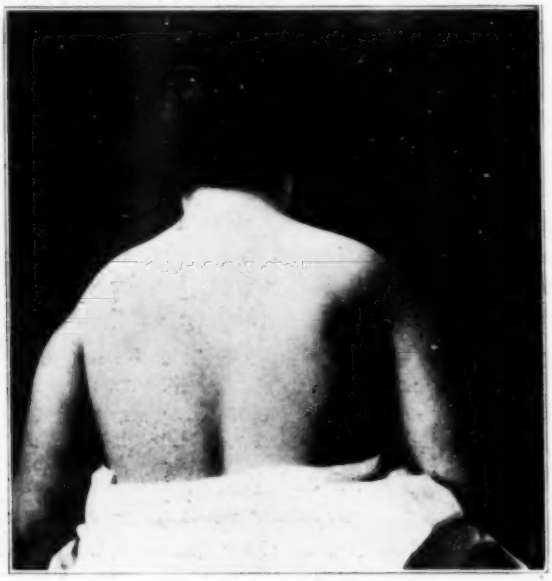


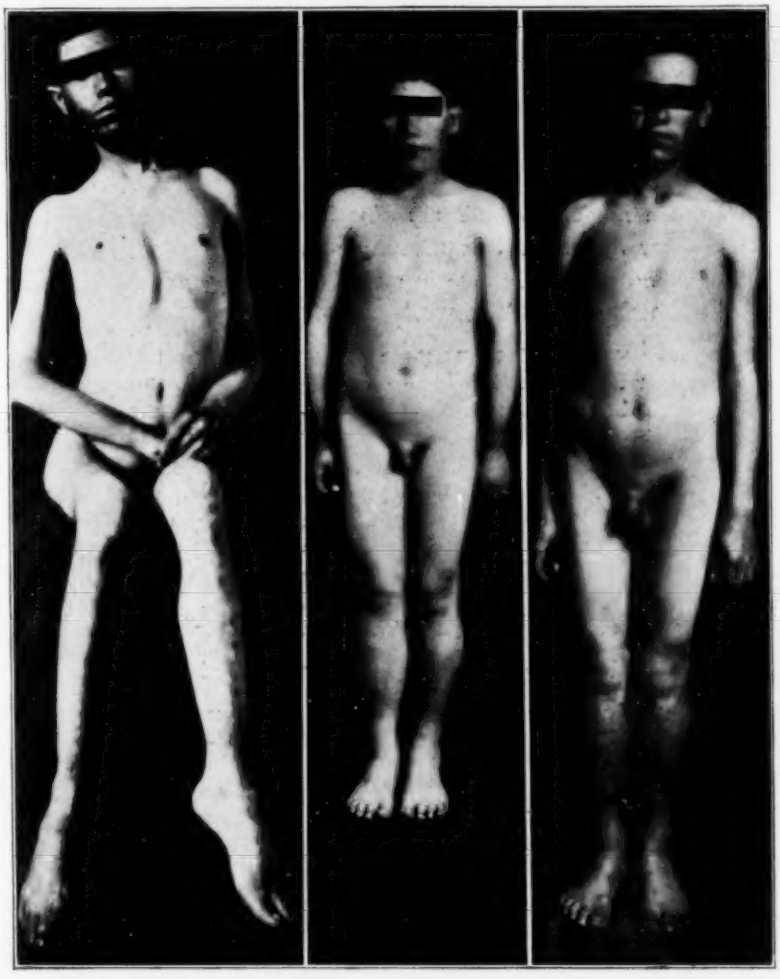
Fig. 1.—Case 2, pseudohypertrophic type.

Family History.—The father of these twelve children died, at the age of 52, from pneumonia following influenza. He was an illiterate farm laborer of Scotch descent, the third of fourteen children. Seven of the family reached maturity; one died when she was 29 during a period of mental excitement. She had had two previous mental attacks. Another sister has had similar periods of excitement. No other evidence of nervous or mental trouble has been found in the father's family. His mother is still living at the age of 75. His father died at 90.

The mother of the twelve children is about 48 years of age. She had typhoid fever at 28, and a recurrence eleven years later. She is the oldest of twelve children, eleven of whom are living and well. Her mother is also living and well. No history of nervous or mental disorder or of muscular dystrophy has been obtained in any member of the mother's family.

Case 1.—John, the second child, died Jan. 19, 1914, from pneumonia at the age of 18. He was afflicted with progressive muscular dystrophy, which, his

mother states, began when he was about 7 years of age. The first indication observed was his inability to lift things up to the table. He gradually lost his ability to run and at the age of 14 was unable to walk, although later he walked for a short time. He gradually lost the use of his arms, but was able to use his hands until the time of his death.



A *B* *C*

Fig. 2.—*A*, Case 3; *B*, Case 7; *C*, Case 5.

Case 2.—Annie, the third child, is 29. Her approximate height is 59½ inches (151 cm.); and weight 145½ pounds (66 kg.). She can neither walk nor stand on her feet; is unable to lift either arm, but can use her hands. She is partially deaf. The patient noted weakness in her back when she was about 5, but walked until about eight years ago. She is very sensitive to cold. She is of the dullard type, having reached the fourth grade when about 14 years of age,

but is normal in personality makeup. She appears to be "fleshy," and one might judge she had good muscular development. She is of the pseudohypertrophic type. The arms and legs are of equal development bilaterally. Some hypertrophy is visible externally, opposite the upper third of the humerus (Fig. 1). No asymmetry of the face is apparent. The fingers are rather short and decidedly tapering, with hyperextension when she straightens out her hand. Power is fair and equal bilaterally, but all the contractile force is exerted by the short flexors. There is marked cyanosis of the face and other parts of the body, and she complains of cold on slight exposure. Sensitivity to heat, cold and pain are normal.

The pupils are equal and regular, and react promptly and equally to light, but the excursion is short. The triceps, achilles tendon and patellar reflexes are lost. The abdominal reflexes are present, but sluggish. The plantar reflexes are both weakly present and of the normal flexor type.

The basal metabolism rate is minus 17.4 per cent.

Case 3.—Taylor, the eighth child, is aged 19. His approximate height is 70 inches (178 cm.); weight 96¾ pounds (44 kg.). He is unable to walk, but has considerable strength in his arms and is able to move his chair about the house. The patient states that stiffness and weakness became evident when he was 7 years of age, and that this was more pronounced in the beginning than in his older brother and sister. He has been unable to walk for five years. He is able to use his hands to make copper "jewelry," shaping copper wire with tweezers.

He reached the third grade in school, and is ordinarily intelligent with a normal personality makeup. He cannot straighten the hip or the knee joint on either side, there being a contraction of about 45 degrees. There is apparently normal abduction and adduction of the thigh and ankle, and toe movements are performed normally. The fingers and hands show weakness of the long extensors. The supinator longus responds very weakly in voluntary movement. Pronation is fairly well performed. The shoulder girdle muscles are much atrophied. There are no definite contractions in the upper extremities. There is decided atrophy of the lumbar, thigh and calf muscles, and a decided foot drop (Fig. 2*A*). On measuring circumferences, little difference can be found between the two sides. There is no evident facial asymmetry. Marked lumbar lordosis is apparent. The circumference of the trunk at the nipple line is 78.5 cm.; at the xyphoid cartilage it is 77 cm.; and at the level of the umbilicus 64.5 cm. There is an apparent prominence of the right side of the chest, which is due to a dorsal scoliosis in addition to the lumbar lordosis.

There is a decided tache cérébrale. The temperature and pain sense and sensitivity to articular movements are normally acute. No radial, triceps or patellar reflexes can be elicited. The achilles, plantar, cremasteric and abdominal reflexes are present and equal on the two sides, but sluggish. The pupils respond both to light and in accommodation. The Wassermann reactions are negative; the blood sugar is 0.1 per cent.; the basal metabolism rate is plus 10 per cent.

Roentgen-ray study of the sella turcica gives no evidence of abnormality. Roentgenograms of the elbow, hand, knee (Fig. 3) and ankle show marked and extensive reduction of density. This may be an osteoporosis due to atrophy from disuse or an atrophy originating in the same manner as, but at a later time than, the muscle atrophy.

Case 4.—Henry, the ninth child, is 16 years of age. His height is 64½ inches (164 cm.); weight 91½ pounds (41.5 kg.). This boy has been unable

to walk for about one and one-half years. He first noted weakness when about 7 years of age, which has gradually progressed. At present he drags himself about with his hands; he cannot creep, being unable to lift his knees and flex his thighs. He has difficulty in raising his arms. When on his hands and knees he is unstable and is very easily turned over on his side, from which position he has considerable difficulty in resuming the hand-knee position.

Fig. 3.—Bones of knee joint in Case 3.

Power is good on each side, although his right hand is stronger than the left. There are no contractions apparent in the upper extremities. Both legs flex completely at knee and thigh, with slight contraction at each joint. There is marked foot drop. There is marked atrophy of the pectoral muscles and of the entire shoulder girdle group, as well as of all muscles of the back and of the thighs. There is a marked cyanosis of both feet and thighs. The skin of the lower extremities is coarse and dry.

The pupils are regular, equal, and react to light and in accommodation. The triceps, radial and patellar reflexes are absent on both sides. The achilles, cremasteric, and plantar reflexes are weak. No abdominal reflexes or ankle clonus can be elicited. Examinations of the blood Wassermann reaction and of the urine are negative. Blood sugar is 0.1 per cent. The basal metabolism rate is minus 1.0 per cent. Roentgenograms of the head reveal no abnormality of the sella turcica.

Case 5.—Orville, the tenth child (Fig. 2 *C*), is 14 7/12 years of age. His height is 60 inches (152 cm.); his weight 87 pounds (39.5 kg.). This lad is still attending public school, being in the fourth grade. He experiences increasing difficulty in walking, and holds his hands out behind him for balance. This weakness began when he was about 7 years of age. He cannot climb stairs without the help of a side rail. He falls down easily and mounts to the standing position in the well-known manner of such subjects (Fig. 4). He has a thickness of speech, and impresses one as being very "careful" in all his actions. He is of the dullard type, having a mental age of 11.7 years on the Yerkes-Bridges point scale.

He has a good grip in either hand. There is a marked weakness of the pectoral, deltoid and scapulo-humeral muscles. He is unable to raise his arms sideways. On sitting and bending forward he cannot resume the vertical trunk position without placing his hands on his knees. In the heel-to-knee test he exhibits marked clumsiness in raising his foot, the awkwardness being the result of muscle weakness. Coordination of movements is not the primary difficulty. When lying in a supine position he is unable to raise either foot straight up in the air by flexing the hip. There is a weakness in the abductors and extensors of the thighs.

Pain and temperature senses seem to be normal. The radial reflexes cannot be elicited. Both patellar reflexes are present, but weak, the right being somewhat stronger than the left. The plantar reflexes are weak, but are of the flexor type. The triceps, achilles, abdominal and cremasteric reflexes are equal and normal on the two sides. The pupils are regular, and react equally and promptly to light and in accommodation.

The blood Wassermann reaction and urinalysis were negative; blood sugar was 0.095 per cent.; the basal metabolism rate was plus 4.6 per cent. Roentgenograms of the sella turcica exhibit nothing abnormal. Lateral roentgenograms of the spine show beginning lordosis with abnormal separation of the bodies of the third and fourth, and fourth and fifth lumbar vertebrae. The roentgenogram of the knee shows normal bone structure, the rarefication seen in Case 3 being absent.

Case 6.—Ira, the eleventh child, is aged 12. Her height is 65½ inches (166.5 cm.); her weight 76 pounds (34.5 kg.). This girl is in the fourth grade in public school. She shows oncoming muscular dystrophy by her awkward and slow movements. Her mother says she first noticed this five years ago. She appears to have good muscular development. The skin below the knees is rough, dry and darkly pigmented, resembling an early stage of pellagra. The skin on the arms and trunk appears healthy. There is a left scoliosis and slight lordosis of the lower dorsal vertebrae. There is a marked inability to climb stairs and also to rise from the reclining and sitting positions. She sits with a bowed back. There is no apparent atrophy of the shoulder girdle muscles, nor winging of the scapulae. There is no apparent foot drop. There is a limitation of flexion of the thighs on the trunk.

Pain and temperature senses, as well as sensitivity to joint movements, show no abnormalities. There is no Romberg sign and no tremor of the tongue or hands. The tongue is large. The biceps, radial, triceps and achilles reflexes are normal and equal on the two sides. The patellar reflexes are sluggish being elicited only on reinforcement. Both plantar reflexes are of the normal type, but are reduced in intensity. There is no ankle clonus. The pupils are regular, equal, and react to light and in accommodation, the right more decidedly than the left.

The blood Wassermann test and urinalysis were negative; blood sugar was 0.089 per cent.; the basal metabolism rate was minus 22 per cent. Roentgenograms of the sella turcica, fore-arm and knee exhibited nothing abnormal.

Fig. 4.—Manner of rising to erect posture; Case 5.

CASE 7.—J. D., the twelfth child, is about 10 years old. His height is 49½ inches (125.75 cm.); his weight 62 pounds (28 kg.). This boy is in the first grade in public school. He shows some difficulty in stooping and in recovering the erect posture. He can, however, stand on his heels. The muscle contraction is equal on the two sides. He runs with the whole side of his body, his hips swaying from side to side, seemingly lifting his hips and legs from the shoulders. These defects of movements indicate a beginning primary atrophy of the muscles of the lower back.

The skin of the calf is brown and dry and has the appearance of ichthyosis. Muscular development appears to be good and no deformity is noted. Figure 2 exhibits the contrast, in this respect, with Case 5. There is no tremor of the fingers or of the tongue; the latter is large. There is no Romberg sign. Heel-to-knee, finger-to-nose, and finger-to-finger tests are well performed. Articular, pain and temperature senses seem normal. No biceps reflexes can be elicited in the arms. The reflexes of the triceps and of the long extensors

of the hands are slightly weak on both sides. The patella, achilles, plantar, abdominal and cremasteric reflexes are normal in intensity and equal on the two sides. No ankle clonus is elicited. The pupils are equal, regular, and react normally to light and in accommodation.

The Wassermann reaction and urinalysis were negative; blood sugar was 0.095 per cent.; the basal metabolism rate was plus 10 per cent. Roentgenograms of the head and knee show no abnormality.

OTHER MEMBERS OF THE FAMILY

There are five apparently unaffected members of this family, two males and three females. The first born is an able bodied, healthy male of 32. He is married and has four apparently healthy children of 11, 8, 3 and 1 years respectively.

Three apparently healthy women of 25, 24, and 22 years, respectively, are the fourth, fifth and sixth, in order of birth. The eldest at the age of 16 gave birth to one child which died in infancy. The next has four healthy children of 6, 4, 2 and 1 years respectively. We saw neither of these sisters, but saw the third normal sister, who has three healthy children of 3, 2 and 1 years, respectively.

The seventh born is an apparently healthy male of 20. His only child, 1½ years old, has recently died of pneumonia.

Of the twelve in the family, five males and two females exhibit progressive muscular dystrophies, being in the order of birth, the second, third, eighth, ninth, tenth, eleventh and twelfth. The oldest girl affected is the only pseudohypertrophic type among the six cases examined. Apparently the brother who died was a case of primary atrophy.

It is of interest to note the general failure of the pseudohypertrophic to conform to the characteristics reported by Gordon [26] when he exhibited a case of pseudohypertrophic muscular paralysis at the Devon and Exeter Medico-Chirurgical Society. Gordon stated that all children of the pseudohypertrophic type die early; in nearly every case at or before puberty. This patient (Annie) is 29; and the next affected sibling is 19. We did not obtain a history of, nor observe, hypertrophy in one of the other cases. Gordon states that contractures are much more marked in the pseudohypertrophic form, but we noticed no contractures in this case of pseudohypertrophy, while in her affected younger brother (Taylor), who has a primary atrophy, contractures are marked. Gordon states that pseudohypertrophic types are generally diagnosable at 6 years of age, while primary atrophic cases are apt to begin much later. All these cases showed first symptoms at the seventh year. He states that five of six pseudohypertrophics are boys and that primary atrophy occurs as frequently in females as males. The case of pseudohypertrophy is in one of two girls affected and all the boys have primary atrophies. This pseudohypertrophic case is one of

26. Gordon, W.: Muscular Dystrophies, Lancet **1**:759 (April 14) 1923.

seven in the family affected with muscular dystrophy and Gordon states that pseudohypertrophic types are apt to present no history of other cases in the family. As there was no proved heredity in this family, we can bring no facts to bear on Gordon's statement that each type (pseudohypertrophic and primary atrophic) tends to breed true. We can note no difference between this pseudohypertrophic girl and her primary atrophic brothers and sister in regard to the muscles affected or the disabilities evident. She seems to suffer from a similar character of atrophy affecting the same muscles, the only difference being that in her case adipose or connective tissue or both have replaced and supplemented the atrophied muscle fibers. Not one of our six cases exhibited atrophy of face muscles.

This group of cases illustrates well the familial tendency of the disease. We find no evidence of direct inheritance of this disability. The insidious appearance of first symptoms, without evidence of the influence of acute infections, and at about the same age in each instance, would suggest some common cause, which becomes active at about the same period of physical development. It has been suggested by Janney and his collaborators [6] that muscular dystrophy may represent a symptom complex due to abnormal functioning of various ductless glands affected singly or collectively. Their studies showed marked decrease (from normal) in preformed creatinin and abnormal presence of creatin in the urine; low values of creatinin and normal amount of creatin in the blood; hypoglycemia; and delayed utilization of glucose.

The lack of available hospital facilities in these cases made detailed metabolism studies impracticable, but the findings obtained would not support the contentions of these authors in reference to the presence of hypoglycemia, as the fasting blood sugar in our cases was within recognized normal limits.

The basal metabolism determinations of these six cases do not point clearly to either hyperfunction or hypofunction of the thyroid gland as the etiologic factors of muscular dystrophy. The findings in the cases herein studied vary from minus 22 per cent. to plus 10 per cent.

STUDIES OF THE METABOLISM IN EPILEPSY

I. THE NONPROTEIN NITROGENOUS CONSTITUENTS OF THE BLOOD *

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Many physicians believe that defects of protein metabolism are of etiologic importance in epilepsy. This belief finds expression in the low protein diet which these clinicians prescribe for epileptic patients. The basis of such a belief is partly the result of therapeutic test and partly the result of laboratory experiment. Many writers have reported the beneficial effect of low protein, or of purin-free diets, though this favorable testimony is not unanimous. Cuneo,[1] for example, states that improvement is more marked with a high protein than with a carbohydrate diet. Weeks [2] et al. find no relation between diet and convulsions. The experimental evidence for faulty protein metabolism is found in the work of numerous investigators, mostly French, English and German, who have reported variations in the amounts of urinary uric acid, urea, ammonia or total nitrogen in the periods preceding or following convulsions. An analysis of these published data is difficult because of the contradictory nature of the evidence presented and because of the variety of the methods of chemical analysis used. Many of the methods are now considered obsolete. In those experi-

* From the laboratories of the Department of Neuropathology, Medical School of Harvard University, and the Monson State Hospital. This research was made possible through a grant by the Committee on Epilepsy, New York City. This paper is No. 6 of a series of studies in metabolism from the Harvard Medical School and allied hospitals. The expenses have been defrayed in part by a grant from the Proctor Fund of the Harvard Medical School for the study of chronic diseases.

1. Cuneo, Gerolamo: Biochemical and Biological Researches on the Pathogenesis of Epilepsy, Riv. sper. di freniat. **45**:408 (Jan. 31) 1922.

2. Weeks, D. F.; Renner, D. S.; Allen, F. M., and Wishart, Mary B.: Observations on Fasting and Diets in the Treatment of Epilepsy, J. Metabol. Res. **3**:317 (Feb.) 1923.

ments in which it was shown that there was decreased excretion of certain products of protein metabolism before a seizure, it has apparently been assumed that there was coincident increased concentration of the product in the blood, which increased concentration was responsible for the convulsion. In addition to this direct experimental evidence, there is the analogy of the convulsions which occur in nephritis and eclampsia, in which conditions unexcreted toxins, doubtless protein in nature, seem to play a rôle.

It occurred to us that knowledge of the subject would be advanced through direct examination of the blood for the concentration of the principal products of protein catabolism. These products are urea, amino acids, uric acid, creatin and creatinin. The sum of the nitrogen of these compounds plus the unidentified, or "rest," nitrogen makes up the total nonprotein nitrogen of the blood. Reliable methods for the clinical quantitative estimation of these substances have been developed only within the last few years.

Obregia and Urechia [3] and Bouttier and Rodriquez,[4] using the old hypobromite method, found no consistent variations in blood urea with reference to convulsions. Bisgaard [5] and his co-workers have reported increase of blood ammonia preceding seizures. Their findings are concerned more directly with the acid-base equilibrium of the blood, and will be discussed in a subsequent paper. Weston,[6] in 1917, examined the blood of ten insane epileptics, choosing those who had no seizure for at least forty-eight hours. No abnormality was found in the concentration of the nitrogenous constituents. Pezzali [7] found no variation from normal in the nonprotein nitrogen of the blood in five cases.

The study here reported was made on a group of 129 epileptic patients. About two thirds of these were inmates of the Monson State Hospital at Palmer, Mass. We are greatly indebted to Dr. Morgan B. Hodskins, superintendent of the hospital, for his cordial cooperation in this research. About one third of the patients were in attendance

3. Obregia, A., and Urechia, C. J.: Les variations de l'urée dans le sang, liquide céphalorachidien et l'urine des épileptiques, Encéphale, **17**:134 (Dec.) 1915.

4. Bouttier, H., and Rodriquez, B.: Etudes sur les variations du taus de l'urée dans l'épilepsie, Ann. de méd. **8**:354, 1920.

5. Bisgaard, Jarlov, and Norvig: Undersogelser over Neutralitetsregulationen ved genuin Epilepsi, Hospitalstidende. **4**:49, 1920.

6. Weston, P. G.: Analyses of Blood of Insane Patients, Arch. Neurol. & Psychiat. **3**:147 (Feb.) 1920.

7. Pezzali, G.: The Blood in Epilepsy, Riforma med. **39**:433 (May 7) 1923.

at the Nerve Out Patient Department of the Massachusetts General Hospital, Boston.

The following are the principal facts concerning the patients whose blood was examined:

Sex.—Fifty-five were males, 74 females.

Age.—The number of patients in the various decades of life were: first decade, 2; second, 36; third, 38; fourth, 34; fifth, 12; sixth and over, 7.

Number of Years Since Onset of Convulsions.—From none to nine years, 34 patients; from ten to nineteen years, 43; from twenty to twenty-nine years, 27; from thirty to thirty-nine years, 13; forty years and upward, 3; not ascertained, 9.

Average Number of Convulsions per Month.—Less than one, 10 patients; from one to four, 49; from five to nine, 36; from ten to nineteen, 20; from twenty to twenty-nine, 5; thirty and over, 4; not ascertained, 5.

TABLE 1.—*Time at Which Blood was Taken*

Blood Taken	After Convulsion No. of Patients	Before Convulsion No. of Patients
Less than 24 hours	20	15
1 to 4 days	31	42
5 to 9 days	26	29
10 to 19 days	22	4
20 to 29 days	6	4
30 or more days	10	3
Information not secured	14	32

Frequency of Petit Mal Attacks.—None, 18 patients; occasional, 83; frequent, 8; not ascertained, 20.

Mental Deterioration.—None, 24 patients; slight, 25; moderate, 48; marked, 24; not ascertained, 8.

History of Status Epilepticus.—Four of the patients gave a history of attacks of status epilepticus; 120 gave no such history; in five cases the facts were not ascertained.

Use of Sedative Drugs.—Thirty-seven of the patients were taking bromids or phenobarbital at the time of the test; eighty-four were taking no drugs, and for eight the information was lacking.

Urine Examinations for Albumin.—The results were negative in seventy-eight patients; from the slightest possible to a faint trace was found in nineteen patients; a trace was present in four cases, and the result was not stated in twenty-eight cases.

Blood Wassermann Reaction.—This was positive in two, negative in 102, not reported in twenty-five.

General Physical Examination.—An examination had been made of each patient, and in none was organic disease of the viscera found. Nine patients had hemiplegia. The great majority of the cases were of so-called essential epilepsy. A few had jacksonian epilepsy. No difference was noted between the analyses of the blood in these two groups. We had about 1,300 patients from whom to select, and chose adults who were having frequent or violent convulsions. Table 1 shows the time when blood was taken for examination with reference to preceding and succeeding convulsions.

In the examination of the blood, the following methods were used: for total nonprotein nitrogen, urea and creatinin, Folin and Wu;[8] for amino-acid nitrogen, Folin,[9] and for uric acid, Folin.[10] Blood was drawn without stasis in order not to interfere with the normal relations of cells and plasma. The results of the individual determinations are expressed graphically in the accompanying charts. All the constituents were not determined for every blood sample. Many of the Massachusetts General Hospital patients had more than one blood examination. The total number of patients whose blood was examined and the total number of determinations is stated on each chart. In the charts each determination is represented by a dot. It was not convenient in many instances to obtain blood under fasting conditions, so that division is made into two groups with reference to the time which had elapsed since the last meal. The ordinate in each chart represents concentration of the substance in the blood, i. e., the number of milligrams of the substance in each one hundred cubic centimeters of whole blood. The abscissa has no significance. The horizontal lines mark the upper and lower limits of normal. With the exception of uric acid and amino-acid nitrogen, to be discussed later, these normal values are practically those gleaned by Matthews[11] from the literature. They are rather liberal limits, such as might be found in a group of hospital "normals." Squier and others[12] give more strict limits for healthy persons as follows: non-

8. Folin, O., and Wu, H.: A System of Blood Analysis, J. Biol. Chem. **38**:81 (May) 1919.

9. Folin, O.: A New Colorimetric Method for the Determination of the Amino-Acid Nitrogen in Blood, J. Biol. Chem. **51**:377 (April) 1922.

10. Folin, O.: A Revision of the Method for Determining Uric Acid, J. Biol. Chem. **51**:187 (Oct.) 1922.

11. Matthews, A. P.: Physiological Chemistry, New York: William Wood & Co., 1920, p. 1007.

12. Squier, J. B.; Brandler, C. G., and Myers, V. G.: Significance of Chemical Blood Findings in Urological Conditions, J. A. M. A. **79**:1384 (Oct. 21) 1922.

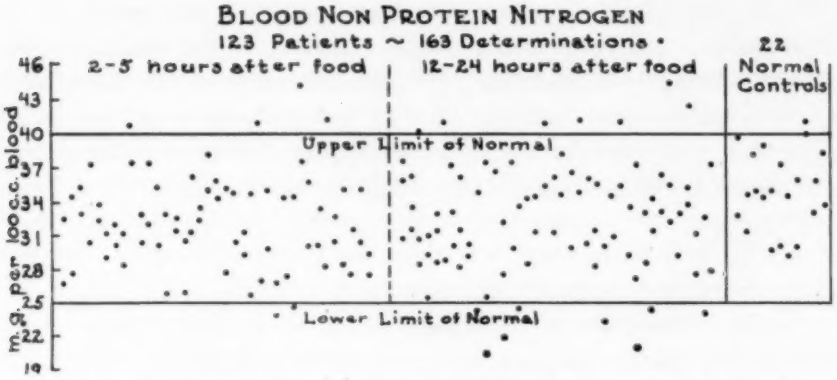

Chart 1.—Determinations of the total nonprotein nitrogen of the blood. In this and other charts, the ordinates indicate concentration, i. e., the number of milligrams of the substance per 100 c.c. of whole blood; the abscissa has no significance. Division is made into two groups with reference to the number of hours after food that the blood sample was taken. Blood from normal controls was taken before breakfast, i. e. from twelve to fifteen hours after food.

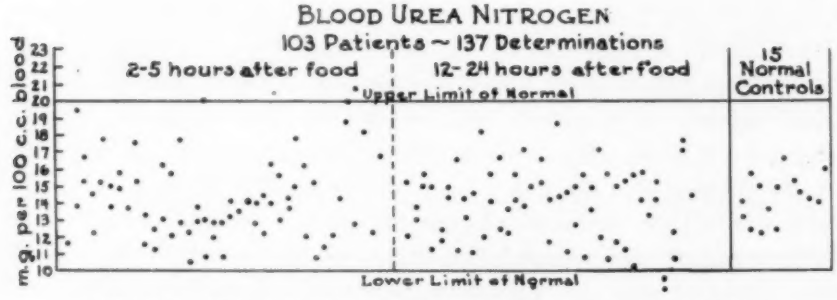

Chart 2.—Determinations of the urea nitrogen of the blood.

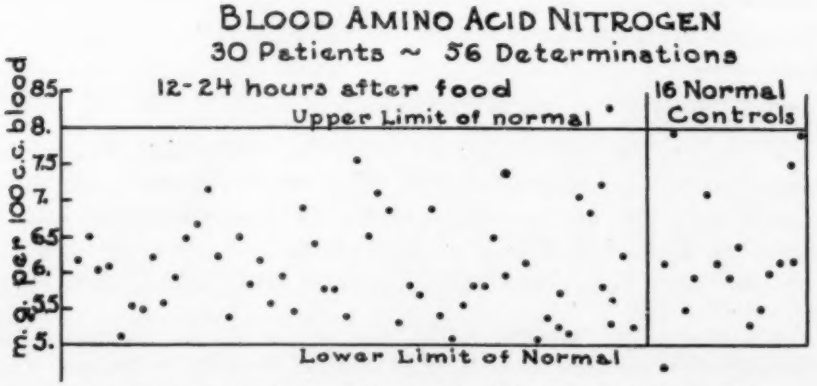

Chart 3.—Determinations of the amino-acid nitrogen of the blood.

protein nitrogen, from 25 to 35 mg.; urea nitrogen, from 12 to 15 mg.; creatinin, from 1 to 1.5 mg. In order to have additional normal figures, which should be at the same time a check on our methods of analysis, we analyzed the blood of about twenty normal persons, mostly medical students. These observations are spotted on the right of each chart.

NONPROTEIN NITROGEN (CHART 1)

The blood of ten patients gave values below 25 mg. These low figures are probably due to the fact that many of the patients were on a low protein diet. Twelve of the determinations were above 40 mg. None of these patients had shown albumin in the urine. It is thought that these scattering high normals have no significance, as they are not markedly higher than our normal controls. The blood with the highest determination (48.7 mg.) showed abnormally high urea nitrogen (30.9 mg.) and uric acid (6.6 mg.); the urine was negative for albumin. At the time blood was taken the patient was unconscious and was having violent convulsions every half hour or so. It was thought that the case was not one of essential epilepsy, for convulsions had begun only two days before and stopped the next day, after an operation for cerebral decompression. At the operation, varices of the pial vessels were observed. Apparently, the high nonprotein nitrogen in this case was the result rather than the cause of the convulsions.

UREA NITROGEN (CHART 2)

Except for the high urea nitrogen figure previously noted, which does not appear on the chart, practically all the determinations were within normal limits.

AMINO-ACID NITROGEN (CHART 3)

The fifty-six determinations of amino-acid nitrogen were made on the blood of patients of the Massachusetts General Hospital. Few normal values for this new method have appeared in the literature, so that we have used the figures found for our own group of normal controls. The blood of patients showed the same range as the blood of the normals. High amino-acid nitrogen is often found in conditions in which there is severe damage to the liver.

URIC ACID (CHART 4)

Determinations of this compound are of particular interest because of the confident assertion of Haig,[13] Krainsky[14] and others, from their

13. Haig, A.: Uric Acid, London: J. & A. Churchill, 1894.
14. Krainsky: Mémoires couronnes, **15**:1901.

observations on urinary uric acid, that convulsions are associated with uric acid metabolism, and follow undue concentration of uric acid in blood and tissues. It will be observed that the upper limit of normal for the concentration of uric acid is placed at 5.5 mg. in place of the usual 3.5 mg. We have found that the Folin-Benedict[9] direct method of uric acid determination gives higher readings than Folin's previous method. Six of the determinations were above 6 mg. All of these were on patients who had been without food for from twelve to twenty-four

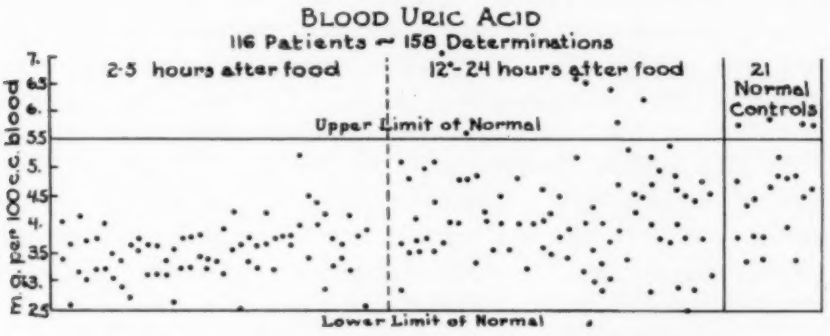

Chart 4.—Determinations of the uric acid of the blood.

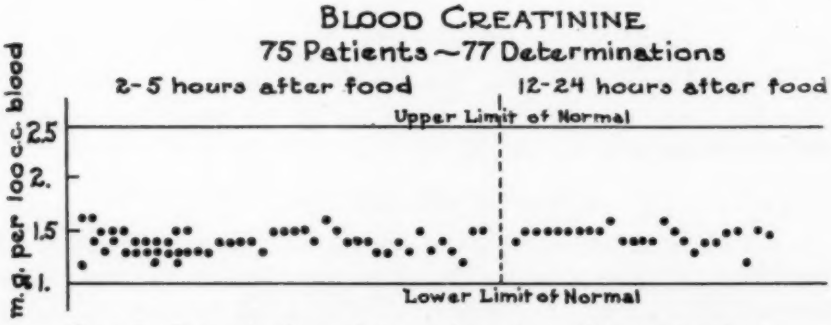

Chart 5.—Determinations of the preformed creatinin of the blood.

hours. From other observations we have made, which will be presented elsewhere, we believe these high values are related to the lack of food and not to epilepsy. Under special circumstances, we secured uric acid concentrations in the blood as high as from 12 to 16 mg.; these high figures were not associated with an increase in the number of convulsions.

PREFORMED CREATININ (CHART 5)

These determinations, as in normal subjects, are monotonous in their uniformity.

UNDETERMINED NITROGEN

In the toxemias associated with nephritis, there is often significant increase in the undetermined or "rest" nitrogen. We did not determine total creatinin, but from the fact that the other known constituents were present in normal amount, we think there was no variation in the amount of undetermined nonprotein nitrogen.

COMMENT

The average values for all determinations are given in Table 2. The number of observations in each case is the number stated on the chart. Inspection of Table 2 shows that the average results for the whole group were remarkably close to the average values obtained for

TABLE 2.—*The Average Values of the Nonprotein Nitrogenous Constituents in the Group of Epileptics and in Normal Controls*

Group	Blood Taken, No. of Hours After Last Meal	Number of Milligrams per 100 C.c. of Blood				
		Nonprotein Nitrogen	Urea Nitrogen	Amino Acid Nitrogen	Uric Acid	Creatinin
Epileptic patients..	2 to 5	32.6	14.2	6.1	3.5	1.3
	12 to 24	32.5	14.4	6.2	4.3	1.4
	All cases	32.5	14.3	6.2	3.9	1.4
Normal controls...	12 to 15	34.9	14.4	6.2	3.7	...

TABLE 3.—*The Average Values of the Nonprotein Nitrogenous Constituents with Reference to Age of Patients*

Age of Patients	Number of Milligrams per 100 C.c. of Blood			
	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin
40 years and over*	33.1	15.1	3.5	1.4
All cases	32.5	14.3	3.9	1.4

* Seventeen cases.

the normal persons. Whether blood was taken under fasting conditions seemed to make no difference except in the case of uric acid.

In order to make sure that figures near or above the upper limits of normal were not due to the elderly patients of the group who might possibly have damaged kidneys, analysis of the figures was made by age of patients (Table 3). Those above 40 years of age showed practically the same average values as the whole group.

It is possible that blood constituents might be normal in the interparoxysmal periods and abnormal for the hours preceding or following a convulsion. Table 4 shows that there was no significant variation in the average figures obtained from blood drawn within twenty-four hours of a convulsion as contrasted with blood drawn more than twenty-four hours before or after a convulsion. Study of the data showed, also,

that there was no significant variation in blood drawn within an hour or two of a convulsion as contrasted with blood drawn from the same patient at an interval of days from a convulsion.

It is yet possible that there would be abnormality for a few seconds only before or at the very beginning of a seizure. Table 5 presents the findings in blood drawn from a patient at frequent intervals and at various times in relation to convulsions, two of the samples being obtained during the tonic stage of a seizure. The variations in the concentrations of the several constituents are small, not more than would be found in successive determinations on blood from a normal person.

TABLE 4.—*The Average Values of the Nonprotein Nitrogenous Constituents With Reference to Whether a Convulsion Occurred Within Twenty-Four Hours of the Blood Taking*

Blood Taken	Number of Milligrams per 100 C.c. of Blood			
	Nonprotein Nitrogen	Urea Nitrogen	Uric Acid	Creatinin
Within 24 hours before convulsion; fifteen cases	36.3	16.6	3.9	1.4
Within 24 hours after convulsion; twenty cases	34.0	15.5	4.3	1.4
All cases	32.5	14.3	3.9	1.4

TABLE 5.—*Determinations of Nonprotein Nitrogenous Constituents with Relation to Convulsions in a Single Case*

Time of Blood Taking		Number of Milligrams per 100 C.c. of Blood			
Day and Hour	Relation to Convulsion	Nonprotein Nitrogen	Amino-acid Nitrogen	Uric Acid	Creatinin
Feb. 27, 9:30 a.m.	4½ hours after 13 hours before	24.0	....	5.50	....
Feb. 27, 1 p.m.	8 hours after 9½ hours before	22.8	....	5.76	....
Feb. 27, 8:30 p.m.	15½ hours after 2 hours before	21.6	5.69	5.56	....
Feb. 27, 10:30 p.m.	During tonic stage of convulsion	22.5	5.40	5.64	1.67
Feb. 28, 12:30 a.m.	1 hour after ½ hour before	24.0	5.25	5.40	1.77
Feb. 28, 2:30 a.m.	During tonic stage of convulsion	22.5	5.11	6.32	1.71
Feb. 28, 8:30 a.m.	4 hours after 12 hours before	24.8	5.39	5.76	1.95

The negative results which have been detailed permit us to make certain positive statements. The finding of normal values for the principal nonprotein nitrogenous constituents of the blood means that in this representative and fairly large group of epileptics there was no impairment of kidney function as concerns excretion of nitrogenous waste products. There was no disturbance, also, of protein metabolism sufficiently gross to show alteration in the concentration of these metabolites in the blood.

It should be pointed out that this study deals only with the substances named. Negative results as regards the concentration of these particular constituents does not mean that in epilepsy there is no disturbance of protein metabolism. There are other normal, or abnormal, products of such metabolism, histamin, for example, which in very small amounts may cause profound physiologic disturbances. The detection of such substances requires other and more elaborate methods of research.

CONCLUSION

In a large group of epileptic patients, examination of the blood for total nonprotein nitrogen, urea nitrogen, amino-acid nitrogen, uric acid and creatinin showed these constituents to be within the limits of normal.

AMBULATORY ENCEPHALITIS

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The number of patients suffering from encephalitis who come to the outpatient department of a large hospital, is many more than might be suspected, and the ease with which they are overlooked is so great that it is necessary to call attention to them. As a rule, these patients pass unnoticed because of the mildness of the disease, and also because of the time that has elapsed between the beginning of the illness and the presentation to the physician. Very frequently the condition is entirely overlooked and only those on the alert for it, or those with a neurologic training, are able to detect the cranial nerve involvement.

During the mild influenza epidemic of this year, I observed a number of such patients. Most of them came to the neurologic dispensary on account of insomnia or peculiar jerkings of the arms. Some of the patients were referred from other departments on account of diplopia or facial nerve palsies. Nearly all of the patients attended to their work until then and the only interruption was when they came for the examination.

I took great care to exclude other diseases, such as syphilis, before reaching the diagnosis, and I took into consideration the sudden onset and other features peculiar to the disease. Most of the patients were able to tell exactly the date of onset, others were able to give a history of a slight coryza, and only rarely was there a patient who was unable to tell the exact date of the beginning of the disease.

One common feature among all these patients was diplopia and insomnia at the onset. As a rule the optician was the first visited, and many patients presented themselves with various shaded spectacles. The other common features were neuralgic pains in the head, face, arms or neck. These pains reminded one of the root pains in spinal cord tumors, or the pains in trigeminal neuralgias. They were described as shooting pains, lancinating, coming on in attacks and lasting a few seconds only. They were always severe and disturbed the patient's sleep. After a day or two they disappeared and the myoclonic movements, which before were slight, became more pronounced. I rarely found a patient with myoclonia who at one time or another did not have the pains.

Patients who suffered from myoclonia also showed an attitude suggestive of a parkinsonian syndrome. The gait, though it did not show loss of the associated movements of the arms, did on the other hand

show a marked slowness of motion. I believe that the ambulatory patients showed a more pronounced tendency toward the parkinsonian syndrome than any other class of patients.

Some of the patients complained of being dizzy, drowsy, or mentally dull. They admitted readily that their ability in the performance of their duties was diminished, that the skill—for example, in handling a needle—was lost, the movements were awkward. They felt tired and weak. One of the patients suffered from mild occupational delirium for weeks before his business associate advised him to consult a physician. Another patient complained that "he had the feeling that he was living in a dream," though he felt the desire for more activity than usual.

It appears that, during this year at least, the myoclonic form is the one prevalent among the ambulatory patients and that a certain grouping is possible. For this purpose I selected from a great number of patients who have come under my observation since January, 1923, fourteen patients. I was able to follow closely these patients and to have under my personal care two who in the course of time developed an acute form of the disease. Of the entire number, three developed acute encephalitis and the rest, as far as could be ascertained, were at the time of the writing of this paper, either in the same condition as when first seen, or slightly improved.

I give here some histories which indicate the characteristics of the possible grouping of the patients as they presented themselves in the outpatient department of the hospitals.

CASE 1.—S. K., a peddler, aged 65 years, came to the Neurological Institute complaining of jerkings of the left arm muscles and neuralgic pains of the same arm, which had been present for three weeks. He kept at work until the morning he came to the institute. At the beginning of the disease, the pain was very severe, so that his sleep was disturbed. He also suffered from diplopia for about four days. The myoclonia started about the same time. He suffered from insomnia and slight headaches, but made no other complaints.

Examination.—The general physical examination gave negative results. The urine examination revealed a slight trace of albumin, but no casts.

Neurologic Findings.—The myoclonus was the outstanding feature of the disease; it involved the muscles of the left arm and the left rectus abdominis. On stretching out his arms, choreo-athetoid movements could be seen involving the left hand. There was no loss of power in the arms and no incoordination. The pupils were slightly contracted and did not react to light or in accommodation. Nystagmus was present in lateral and upward movements. There was a left external rectus and also a left facial weakness. Loss of upward gaze was pronounced. The tongue protruded in the midline. The deep reflexes were all present and normal; the superficial reflexes were present, with the exception of the left abdominal reflexes, which were practically lost (occasionally elicited). There was no Babinski sign and no clonus. The associated movements of the arms were present when walking, but were very slow. The patient tired easily during the examination.

Comment.—This examination was made three weeks after the onset of the disease, and it is impossible to say just what the condition had been at the beginning.

Case 2.—H. G., a messenger boy, aged 19, was referred to the Neurological Institute by Dr. Max Bresler, with the diagnosis of possible encephalitis. He had been ill for four weeks before coming to the institute. The illness started with headaches, dizziness, and insomnia. He had diplopia for a few days, but no myoclonia. After a lapse of about ten days he became very drowsy, which condition passed two days later. Afterward he was depressed, could not work, kept his head in a rigid position and walked with the body bent forward.

Examination.—Intelligence tests showed that the patient was a low grade moron. On inspection, a slight myoclonia could be seen involving the left arm muscles. The pupils were fixed, and a slight nystagmus occurred on lateral movements. The right sixth and third nerve muscles were paretic. Upward gaze was good. There was slight exophthalmos, but no enlargement of the thyroid. On standing he had a typical parkinsonian face and position. On walking there was a loss of the associated movements of the arms. His speech was slow and monotonous. There was marked myasthenia with slight rigidity of the arms, and the entire attitude of the body was that of rigidity. The deep and superficial reflexes were all present and normal.

Comment.—This case was of interest on account of the absence of what could be called an acute stage of the disease, unless we consider the few days of the onset as the acute stage. He developed the parkinsonian syndrome without any acute symptoms. He also showed a beginning psychosis, manifested by depression and decreased psychomotor activity.

Case 3.—L. G., a girl student, aged 19, came to the Neurological Dispensary of the Mount Sinai Hospital, because of a left facial palsy. Two weeks previously she had suffered from diplopia and consulted her physician, who sent her to an optician. Glasses were fitted and the diplopia disappeared. She then had headache and again consulted her physician, who discovered the left facial paralysis. She came to the dispensary for massage.

Examination.—There was no myoclonia. The gait was normal, with preservation of the associated movements of the arms. The face appeared somewhat masked. The pupils were equal, but showed practically no reaction to light or in accommodation. The ocular fundi were normal. The left third and sixth cranial nerves were paralyzed. The left facial nerve was paretic. Upward gaze was good. Tremor was present in the fingers of both hands. The reflexes were all present and normal, and otherwise the examination gave negative results.

Comment.—In spite of the few findings at the examination, this patient gave the impression of being acutely ill, and she was referred to the hospital for admission.

Case 4.—A girl of 12, seen at the Neurological Institute with Dr. Carrol, came in with the diagnosis of chorea minor. She had been ill for three weeks and the illness had started suddenly. She had had a slight diplopia for about two days, but this fact was brought out by the physician questioning her. The chorea was the outstanding feature, except the headaches and the insomnia.

Examination.—On closer observation the choreic movements were really a myoclonia of a severe form. Choreo-athetoid movements were present and involved the left hand. The pupils barely reacted to light or in accommoda-

tion. There was a definite third cranial nerve palsy on the right and also a slight facial palsy on the same side. Nystagmus occurred with lateral movements. The reflexes were all present and there was no Babinski sign nor clonus.

Comment.—At the time of the examination the temperature was 100. It was impossible to say whether we were dealing with the beginning of a serious condition, or with a subacute one. Unfortunately, I lost track of the patient.

From the examinations and observations made, it is important to ask ourselves the question: Did we, in these patients, deal with the beginning of an infection, which abruptly limited itself and established a cure, or did we deal with a very mild—so-called subacute—condition? In face of the experiences of various neurologists this question is important.

Strauss and Globus [1] reported a number of so-called subacute cases, among which there was one having considerable bearing on our subject. It is the case of a patient who suffered from vertigo for a period of six months before the acute encephalitis developed. He finally died from the acute disease. Dr. Isadore Abrahamson, of New York, presented a patient at a conference of the Neurological Department of the Mount Sinai Hospital on Feb. 5, 1923, who had suffered a mild attack of encephalitis two years before, and after two years of apparent health began to develop a parkinsonian syndrome. Recent French literature abounds with the so-called "encéphalite tardive." It is therefore impossible to say whether the mild encephalitis of the ambulatory patient is not the beginning of a long continuing process, which may at any time under favorable conditions develop acute exacerbations.

The following instructive case history will show the importance of the last sentence:

Case 5.—M. S., a designer, aged 42 years, was taken ill with severe pains in the left frontoparietal region, which lasted a few days only and then started in the neck and arms. They disappeared after a few hours, started in the fingers and finally disappeared entirely. He had no diplopia at that time. I saw the patient then. On examination he showed a marked myoclonus involving the left rectus abdominis. Nystagmus was present on rotation of the eyes in all directions. The pupils were fixed. There was definite left third and left sixth nerve paralysis. Loss of upward gaze was very marked. The deep reflexes were present and normal, and of the superficial reflexes the left abdominals were lost. There were no other abnormal findings. Two days later the patient cancelled his appointment with me, as he felt well and was able to leave his house. He was apparently well, and was able to attend to work for two weeks, when he was again taken ill with diplopia and vertigo. I saw him again one week after this second onset. At this time he was semilethargic, suffered from occupational delirium and had all the evidences

1. Strauss, Israel, and Globus, J. H.: Arch. Neurol. & Psychiat. **8**:124 (Aug.) 1922.

of an acute encephalitis. On examination, the findings were exactly like those at the first examination, with the exception that the temperature was 104 F., respiration was 36 per minute, and he was in a semicomatose state, changing into delirium and restlessness.

This patient had therefore what at first looked like a mild attack of encephalitis, which subsided and appeared again after three weeks in the form of an acute and very severe attack.

I had a similar experience with the patient in Case 1 reported above. Two weeks after my first examination I was called to his house, as he was irrational. He suffered from a severe occupational delirium, was very restless and had a temperature of 101 F. This time the myoclonus was very pronounced and involved the entire left side of the body. The deep and also the superficial reflexes were lost. Speech was indistinct, and from time to time he had impairment of breathing. He vomited and occasionally had dysphagia. It took, therefore, five weeks to develop the acute condition from what appeared at first to be a mild attack.

COMMENT

From the study of my cases and from what I gather from the literature, it seems that what is conceived as a mild attack, as an ambulatory case, may in reality be the stage of invasion of a very serious illness. I found no clinical facts from which could be foretold the probable development of a case. Moreover, clinically, the difference between a mild and a severe case does not seem to be in the findings at the examination. As a matter of fact, a mild case may show more cranial nerve involvement than a severe case. As can be seen from the cases reported above, those patients who developed a severe form of the disease, showed no more findings then than at the beginning of the disease.

I will, however, mention a few points which in my opinion are possible indications of a severe form, or indications that a mild case is on the threshold of the development of an acute condition.

I noticed that *those patients who showed a loss of the upward gaze developed the severe form of the disease,* even if weeks had passed from the time of onset of the illness. *Of patients who were acutely ill, those who had showed a loss of the upward gaze were in a more serious condition than the others.* The fourth nerve affection was the last to recover, and could serve as an indication of the patient's condition.

Another important point was the loss of the abdominal reflexes, which I confess I did not find in last year's cases. These reflexes may be lost in the face of very lively deep reflexes, and of diminished or lost deep reflexes.

CONCLUSIONS

1. A mild encephalitis is a potential severe case. The so-called subacute encephalitis possesses the danger of an acute exacerbation.

2. The loss of the upward gaze is an indication, when found in a mild case in an ambulatory patient, of the eventual development of an acute and possibly severe condition.

3. Patients in Group 2 should be considered as acutely ill and should be treated as such.

CONSTITUTIONAL PSYCHOSES ENDING IN PERMANENT RECOVERY

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Some time ago one of us offered a theory of personality based mainly on psychiatric experience.[1] In dealing with the problem of analysis of personality it was pointed out that the task of such analysis consisted not only in direct observation of manifest traits, but also in bringing to light possible latent ones. Among the means for accomplishing the latter, a more systematic utilization of ontogenetic data was suggested.

In that theory five elementary components of personality were distinguished, namely: normal, antisocial, cyclothymic, schizophrenic, and epileptic. Each of these was considered to be determined by special germ-plasm factors, and some evidence was brought forward which seemed to indicate that these factors were ranged in a scale of dominance, the normal factor being epistatic (in Bateson's sense) in relation to the others, the epileptic factor hypostatic, and the remaining factors epistatic in relation to some and hypostatic in relation to others, their positions in the scale of dominance being as given in the above enumeration.

The point was made that all the personality components of an individual are not present at birth or in infancy, and that they do not all make their appearance simultaneously. To quote: "Every trait has its own ontogeny, and before the final blending takes place there is considerable opportunity for analytic study. In the case of a given trait, the time of its appearance, the intensity of its manifestations, and the permanence of its persistence are probably dependent in part on the quantity or dosage of its germ-plasmic determining factor and in part on the development of other factors occupying an epistatic position in the scale of dominance."

Attention was drawn to the fact that traits which, when observed in grown persons, are recognized as neuropathic, are more or less regularly present in children, later to be overlaid, brought under control, and finally rendered latent as the epistatic "normal" factor reaches maturity.

The present study was undertaken with a view to further testing the validity of this theory. As far as epilepsy is concerned, it is not necessary to seek new material, for the fact of permanent recovery, with

1. Rosanoff, A. J.: A Theory of Personality Based Mainly on Psychiatric Experience, Psychol. Bull. **17:** (Sept.) 1920.

or without treatment, from epileptic manifestations in childhood or youth is within common experience. In dealing, however, with the psychotic elements of personality—the cyclothymic and schizophrenic—it should be borne in mind that although manifestations of these factors, more or less pronounced, are often observed in childhood, yet fully developed psychoses are seldom seen in individuals under 15 years of age.

For instance, the statistics of the New York state hospitals show that during the fiscal year ended June 30, 1920, there were, among first admissions, 882 cases of manic-depressive psychoses and 1,926 of dementia praecox. Of these only six and five, respectively, were in patients under 15 years of age. The maximum incidence was reached in the 25-29 year age group, which is represented by 154 manic-depressive and 409 dementia praecox cases.

It would seem then that, even in subjects who are destined to become state hospital patients, the ontogenetic development of psychotic traits is not completed as a rule until early adult life. In childhood such traits exist generally in rudimentary or immature form.

Theoretically, the cases in which rudimentary psychotic manifestations are seen in childhood have at least three possibilities before them: (1) the "normal" constitutional factors mature early, causing the psychotic factors to fade before the age of incidence for fully developed psychoses has been reached; (2) the "normal" factors are largely wanting and the subject, as he matures, develops a psychosis which is either recurrent, or chronic, or deteriorating in its course; (3) the "normal" factors are relatively late in maturing, thus permitting the psychosis to develop; such psychosis, however, ends in permanent recovery as full development of the "normal" factors is eventually attained.

Instances illustrating the first two of these possibilities are common enough in the experience of every psychiatrist. Instances of the last mentioned possibility—that of permanent recovery—do not come to attention as such in the psychiatric clinic, for the reason that a patient, following recovery and discharge, is not seen again at the clinic, except in the event of recurrence, and his further course is not recorded.

It has thus come about that current psychiatric classifications take scarcely any cognizance of the permanent recoverability of the constitutional psychoses. The general assumption seems to be that the outlook in a given case is either for recurrence, chronicity or deterioration.

The bulk of the material of every clinic strongly supports this assumption. The evidence against it—cases discharged as recovered and subsequently not readmitted or otherwise heard from—is obviously inconclusive; for whether these are cases of permanent or only temporary recovery could be known only through follow-up investigations after the lapse of many years—an investigation which, as far as we

know, has heretofore not been undertaken; the present study represents such an investigation.

We have assumed that the "normal" germ-plasm factor in some cases does not attain its full development until the middle or latter part of the third decade of life. The assumption of this late period for the completion of development might be considered a weak point in the theory. The answer would seem to be that no age can be given as one of complete general ontogenetic development, some traits maturing early and others late. In some individuals, for instance, the depth of hair pigmentation continues to increase far into the fourth decade. A more striking instance of a late maturing trait is that of Huntington's chorea, the first manifestations of which generally appear in the late thirties and sometimes not earlier than the fifth decade of life, i. e., when, for some traits, senile involution has already set in—atrophy of hair pigment, cessation of ovulation, etc. It would seem, therefore, that the assumption that the "normal" germ-plasm factor does not mature in some cases before the middle or late twenties does not necessarily invalidate the theory.

THE MATERIAL

The material for this study was found among the admission records of the Kings County Lunatic Asylum, Brooklyn, N. Y. (now Brooklyn State Hospital), for the years 1880 to 1889, inclusive.[2] These records were gone over in chronological order and records were selected of patients who had been admitted at an age not over 25 years and who had subsequently been discharged as recovered. Cases which were later readmitted were then eliminated, and an attempt was then made to secure by means of field investigation the subsequent histories of the remainder.

As we had expected, it proved in most cases impossible to find these former patients or their relatives or friends. In some cases, in which we succeeded in tracing their present whereabouts, the investigation had to be abandoned for lack of cooperation or for some such reason.

In eight cases we were successful in securing data concerning the life of former patients up to the time of this study (1921-1922). These former patients had attained ages of from 50 to 65 years; and they had been free from psychosis for periods of from thirty-one to forty years. Abstracts of their clinical histories are submitted herewith.

REPORT OF CASES

CASE 1.—*Psychosis, probably of manic-depressive nature, at the age of 25, with recovery in twelve months. Death at the age of 65 without further nervous or mental trouble.*

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2. We wish to thank Dr. Isham G. Harris, Superintendent of the Brooklyn State Hospital, for granting us access to this material.

H. P., a woman with a family history that was unimportant, was born in Ireland, received a common school education, and came to the United States when a young girl. She was jolly and fun loving by temperament. She married a German at the age of 22 and her married life was said to have been happy. She was very happy when she learned that she was pregnant and was well during the pregnancy; the baby was stillborn.

About a week after confinement she became excited and was committed to the hospital, Sept. 29, 1880, the papers stating that she had been very much excited, had assaulted her husband, had said her right hand was dead, and had talked constantly and incoherently in a low tone, although at times she answered questions intelligently. Her temperature was normal. The hospital records state:

Oct. 26, 1880: Yesterday the patient asked for a knife with which to kill herself; this morning she denies this. She talks about having been married frequently and mentions the names of many men. Her conversation is wandering and flippant.

Oct. 30: Walks up and down the hall and tries to get out; becomes abusive and will not eat or take her medicine.

Jan. 5, 1881: Mental condition unchanged. At times attacks other patients.

March 4: She has a handkerchief tied loosely about her neck and when asked what that is for she at once became flushed and agitated and showed by her manner that she was not well.

Sept. 10: The patient has gradually become more rational and now behaves quite naturally. Discharged as recovered.

On discharge the patient returned to her home in Brooklyn where she lived until fourteen years ago, when the family moved to Astoria, Queens County. She has since had six children, two of whom died of "summer complaint" in infancy, while four are living.

Since discharge the patient has been calm and serene, never nervous or worried. She has kept house and lived an average life with her husband and children. In March, 1920, she became ill and died the following August; but the nature of the illness and cause of death have not been ascertained. She remained free from psychotic symptoms during this illness.

CASE 2.—*Psychosis, probably an acute schizophrenic reaction, at the age of 20 with recovery in three months. No return of psychotic symptoms to age 60.*

J. H., a woman, aged 20, was admitted July 17, 1881. The family history was negative except that the father was said to have been of emotional temperament. The patient was strong and healthy as a child, but quiet and retiring, never played with other children, and later never went out with boys. She attended high school and normal school, was very conscientious, and studied from seven in the morning until eleven at night. She was always very religious; never went out or enjoyed the usual good times of young people. She was very ambitious.

The psychosis developed gradually. She had hoped to become a school teacher, against her parents' wishes. One of the city aldermen encouraged her to continue her studies and promised to secure a position for her in the city schools on graduation. She worked hard to qualify, and on doing so failed to get an appointment. She was greatly disappointed, became despondent and seemed to lose interest in life. She heard voices threatening to injure her, and developed a sexual trend of ideas; she told her physician that she was

disturbed a great deal by an overpowering sexual desire but felt it would be wrong to gratify it unless married.

The commitment paper and clinical records state that the patient remained in any position for a long time without moving in the slightest. Her countenance was much dejected, and she shed tears without apparent cause. She had hallucinations of hearing, and imagined that she heard people coming to kill her. She had a marked tendency toward suicide. Aug. 4, 1881, she was improved but still depressed. Oct. 5, 1881, it was noted that she had shown gradual improvement since admission, was very quiet, and this day was discharged as recovered.

The subsequent history is as follows: After discharge from the hospital she entered normal school and graduated with high honors. Henry Ward Beecher was present at her graduation and was so impressed with her ability that he secured for her a teacher's position in the Brooklyn schools the following day. For twenty years thereafter she taught successfully. She then entered a convent, where she gained rapid advancement and today she holds the position of Mother Superior.

She is described as reserved and dignified, but very active, alert, and a woman of the world. She is a splendid executive, said to be just, firm, never wavering in her decisions, and never changing her mind after it has once been made up. She is purposeful and business like and presents a marked contrast to the meek timid type of person that she was in her girlhood.

CASE 3.—*A manic-depressive psychosis, at the age of 17, with recovery in six months. No recurrence of psychotic symptoms to the age of 50.*

K. M., a woman, aged 17, was admitted June 11, 1888. The paternal grandmother and a cousin have been insane, the latter being then a patient in a state hospital; a dead sister had been silly, childish, weak and easily led. The patient, born in 1871, was healthy as a child, and did well in school though she left before graduation to go to work. She was employed in a dry goods store until the onset of the psychosis. She was always lively, full of fun and enjoyed the company of other girls and boys.

During the severe storms which occurred in March, 1888, the patient was exposed to the cold and her menstruation ceased. Otherwise she seemed well until the latter part of April, when she complained of feeling badly, went to bed, and seemed very quiet. On May 10, the relatives first noticed symptoms which they recognized as mental: the patient imagined she heard roosters crowing about her; pulled at her hair and struck her head against the wall; tried to stuff her apron down her mother's throat; at other times was quiet and conversed only on being persistently urged, when her utterances were not coherent and her mother could not understand her. The commitment paper states that when questioned, she gazed vacantly and gave no answer. Sometimes, when persistently questioned, she replied in monosyllabic whispers. She refused to take food or drink, sat for hours in one position, and paid no attention to what was going on about her.

In September she began to improve, occupied herself with sewing, grew more communicative, and told her physician that she had not wished to converse with him as the Lord had told her not to speak. On October 14, she was paroled to her mother and on December 2 she was discharged as recovered.

At the age of 18, less than a year after her discharge from the hospital, she married; she has since had four children who are living and well, aged

32, 26, 22 and 11 years, respectively. She had had no return of the psychosis. She is now rather frivolous, negligent about her household, gets up late, dresses flashily, goes to shows or movies frequently in the afternoon; she drinks moderately as a rule but occasionally to the point of intoxication, when she becomes jolly but not disturbed.

CASE 4.—*Psychosis, apparently a mixture of manic-depressive and schizophrenic manifestations, at the age of 24 with recovery in one month. At age 57 normal and well balanced.*

M. J. M., a woman, aged 24, was admitted Feb. 23, 1889. The family history was negative except that a sister seems to be of rather low grade mentally, and a brother died of brain fever at the age of 3½ years. This boy had been walking in the dark, when a cat brushed against his bare feet and frightened him; he was sick for one week, during which he talked of the cat almost incessantly.

The patient was born in Brooklyn, in 1865, was healthy as a child, graduated from grammar school at 14, then worked for seven years, and married at 23. Her temperamental make-up seemed normal. She has had eight children, five of whom are living and normal; one is described as very quick tempered; another has dementia praecox and is now a patient in a state hospital; another died in infancy, in convulsions that were attributed to teething.

The first child was born in the early part of February, 1889. Delivery was normal and the patient was in labor only a short time. On the third day of the puerperium she was awakened from sleep by a sudden noise which frightened her so that she jumped out of bed. She was alone in the house at that time and managed to get back to bed, but immediately developed psychotic symptoms. She said she had been conversing with God who told her that her aunt had stolen her sister's cloak. She turned against her nearest relatives, said her husband was untrue to her, and her father wanted to harm her. She slept poorly; took but little food, saying some one wanted to poison her and her baby. She became quite disturbed and was sent to the hospital three weeks after the onset of the psychosis. The commitment paper states: "Believes that she has been in heaven and the Lord spoke to her and gave her His hand. Says she did not see the Lord. Says also that she hears angels singing at night in her room. She is noisy and violent. Screams and talks at night. Declares that the nurse is trying to poison her, refuses food."

On admission to the hospital, the patient was excited, running aimlessly about the hall; at times she said the physician was a priest, at others that he was her husband. She would not eat and had to be fed by tube. A few days later she began to improve and a note dated March 10, 1889, states that her behavior had become normal; the delusions and hallucinations had disappeared. She was discharged as recovered March 17, 1889.

Since discharge she has given birth to seven children and has been entirely free from any psychotic trouble. Seven children are now living, ranging in age from 19 to 31 years, five of them free from nervous or mental trouble, the facts with regard to the other two having been already mentioned. Today the patient is strong, healthy and active. Her husband died several months ago and she has since conducted his fish business with the aid of her eldest son. When interviewed in the course of this study, she made an excellent impression, seemed sensible, intelligent, well balanced, and expressed herself well; she cooperated well, was pleasant, friendly, hospitable, and her appearance was neat and tidy.

CASE 5.—*Brief period of mild depression during pregnancy at age 22; very brief excitement one week after confinement; excitement, apparently of manic-depressive nature, on the fifth day of pneumonia four months later, which lasted about ten weeks and terminated in complete recovery. No recurrence to age 57.*

A. T., a woman aged 22, was admitted Oct. 31, 1887. Her family history was unimportant. The patient was born in New York in 1865, was a healthy child, active and enjoyed playing with other children. She attended parochial school and got along well, but at the age of 13, when in next to the highest class, left, as she wanted to go to work. She worked on artificial flowers for several years and helped her mother at home. She went around a great deal, was sociable and happy by temperament, and had good times at parties. She married an electrician at 20 and the husband was good to her.

The first evidence of psychosis appeared simultaneously with her first pregnancy about three months after marriage. She became melancholy, thought her best friends were against her, did not want to go out. The first baby was born a little less than a year after marriage. Childbirth was comparatively easy and she seemed to be doing well, but about a week afterward she became excited for a period of scarcely more than a day. From this she recovered fully, and for four months thereafter was well both mentally and physically and was able to nurse her baby. In the latter part of September, 1887, she contracted pneumonia and on October 3 developed another attack of excitement. An attempt was made to treat her at home, but after four weeks she had to be committed. While under treatment at home she spent days and nights without sleep, required two attendants because of her violence, had to be restrained, used bad language, seemed not to recognize her nearest relatives, would not take medicine, and was fed with difficulty. The commitment paper states: "She has been for weeks violently delirious and extremely incoherent, laughing and crying alternately and causelessly."

On admission the patient was "noisy and so rambling and incoherent in her talk that little could be understood of what she said." She talked continually about being married to Dr. H. (her private physician), refused medicine and ate poorly. Five days later she was still laughing, talking, and singing continuously, refusing medicine, and requiring persistent urging to eat.

Nov. 12, 1887, she was taken home by her husband, though still unimproved, and recovery followed about a month after discharge from the hospital.

In a retrospective account the patient stated to the field worker that on admission to the state hospital she realized where she was, that the other patients were insane, and everything that had happened. She was so desirous of leaving the hospital that she prevailed on her husband to take her home at the end of two weeks, although very little, if any, improvement had occurred.

Since recovery, she has given birth to eight children, the last when she was 39, eighteen years ago. Five of her children, two sons and three daughters, are living and well, ranging in ages from 20 to 32 years. The first child, a daughter, died at 12 months of "summer complaint" a few months after patient's recovery from the psychosis. The third child, also a daughter, died at 4 months of whooping cough. The sixth child, also a daughter, one of twins, died three weeks after birth. The ninth child, her last, a boy, died when a few months old, also of "summer complaint."

The patient has had a great deal of financial trouble in her life. For many years her husband was unsuccessful in getting a steady position. She

had much occasion to worry about the prospect of securing for her children a proper bringing up and education. She was also saddened by the illnesses and deaths of four children and the deaths of her parents, brothers and sisters. Her work at home was burdensome and unremitting and she had other difficulties and disappointments. Yet she has had no recurrence of psychotic trouble during the thirty-five years that have now elapsed since the recovery from her attack at the age of 22.

The patient is now 57 years of age, but appears much younger. She is well physically, is active and alert, does her own housework, has a pleasant home in a good residential part of the city, is very jolly and fun loving, "as ready as a girl for a good time," and says she never intends to let herself grow old. She impresses one as normal in every way.

CASE 6.—*Very brief psychotic episode at age 17; manic-depressive psychosis at age 19 with recovery in four and one half months; no recurrence up to age 58, although patient is "nervous."*

H. B. M., a man aged 19, concerning whose family history little was ascertained, was admitted Dec. 12, 1882. His grandmother (paternal?) was said to have been "a highly hysterical person and of a very imperious and haughty manner." The patient was born in New York in 1863, was considered normal and lived a healthy boy's life in what was probably a good home. He did well at school, liked the company of other children and got along well with them. He was ambitious and when he reached early manhood worked hard.

Relatives attributed his psychosis to overambition. He was disappointed at not being accorded the distinction and places which he believed he merited, such as election to office in societies to which he belonged, and advancement in business. His disappointment was increased by the circumstance that he wanted to get married and could not do so without making faster progress. His parents thought he was unreasonable in the matter, as he was but a boy, and in the natural course of events could not or should not expect such promotions and preferments.

The first evidence of psychosis appeared at the age of 17 when he had an excited episode which was referred to as an hysterical attack; he wanted to run out of the house; the next day he was all right again. He remained well until Oct. 15, 1882. He was then 19 years of age. At that time, under the circumstances described above, a more severe attack occurred. His conversation became rambling; he said some people were trying to injure him by poison and violence; he talked incessantly; at times he was irritable and aggressive, at others elated and expansive and at others depressed and lacrimose. At times he was so restless and excited as to require sedatives. He said he had discovered the world was round and thus saved it from destruction. He complained of a pain under the left rib, where President Garfield had been shot. Often he would say that he could not breathe and he would throw all the windows open in the house in bitter cold weather. He broke up things around the house and was finally committed. The commitment paper states: "Imagines he has been poisoned several times; incoherent in conversation; mistakes identities; restless and very excitable; at times quite noisy, needs restraint. Says his mother does not know when he was born, because he was born after twelve o'clock at night. Cries and becomes profane and violent."

On admission he was excited, would pick fights with other patients and required sedatives. He walked hurriedly up and down the hall, sang, whistled,

talked a great deal in a rambling way about politicians, President Arthur, railroads, soldiers; sobbed violently for several minutes, then the tears would be checked as suddenly as they began. He became calmer in a few days, but on December 21 was again disturbed: "Talks wildly about licking everybody in the room, his inventions, telegraphs, railroads. Says two of the patients are Chinese, but gives them English names. Bursts into sudden fits of crying. Said: 'There was no crown in Portugal or Spain because I was the first Alphonso man and so the articles were signed.'"

The psychotic symptoms continued, with more or less fluctuation, until about the middle of February, 1883, when definite improvement set in. The patient soon developed insight, was paroled to his father March 4, and March 13 was discharged as recovered.

It was impossible in the course of this investigation to secure an interview with the patient as relatives feared that discussion of his psychosis might upset him. His brother, however, told us that shortly after discharge from the hospital the patient married and his married life has been congenial. The patient is now in the building construction business, has been very successful, is prosperous, and lives in a good residential section of Brooklyn. He is 58 years of age and has been free from recurrence for nearly forty years. Although he is considered a very nervous man, he is quite able to withstand hardships and disappointments. His daughter died recently and he was deeply affected by her death, but has shown no signs of breakdown.

CASE 7.—*Mental deficiency complicated with a psychosis at the age of 22 years, apparently of manic-depressive nature, lasting about eight months. Recovery. No recurrence to the age of 60 years.*

K. B., a woman aged 22, admitted Sept. 17, 1883. Her father was verbose and rambling in conversation, very emotional, and suffered from occasional nervous breakdowns that caused him to give up work temporarily. A paternal uncle was in a hospital for mental disease; and a sister was "highly emotional, almost hysterical" on the occasion of patient's commitment. The patient was born in Brooklyn in 1863, developed well physically, but was backward mentally. She learned to walk when nearly 4, and began to talk about the same time, though has never talked plainly and still has a speech defect. She went to school for several years, but made poor progress and a younger sister soon passed her. She was then taken out of school and an attempt was made by an older sister to teach her at home, but it was long before she learned to tell time by the clock. Temperamentally she was good natured, industrious and easily managed. She never married. Owing to the mental deficiency she was kept at home and did not seem to crave the company of young people. At the age of 20 a position as nursemaid was secured for her, later one as waitress in a boarding house. Her work was satisfactory until interrupted by the development of her psychosis.

The psychosis developed in May, 1883, while she was working as waitress in a boarding house. The work was somewhat harder than she had previously done and the wages were a little higher. This may have had something to do with her breakdown. The relatives stated, however, that a circus came to town and the circus people came to stay at the boarding house where she was employed; the excitement upset her. Nothing else happened as far as is known. She became confused, forgetful and could no longer do errands. She got lost in the streets and said her employer had taken all her clothes. She was then taken home by her father.

At home she seemed dazed, could not do work about the house; she had to be dressed and undressed, or she would put on all the clothing she could find; she would lie in bed muttering to herself all night; at times she had paroxysms of crying and screaming without apparent cause; she kept wondering whether she was married or not. As she failed to improve at home she was finally committed. The commitment paper states: "She does not comprehend the true purport of questions. Continually mopes around the room in a bewildered manner. Occasionally cries and weeps bitterly giving no reason therefor. Incapable of any connected mental effort. Can answer questions only in monosyllables. Sits with face averted."

After admission she remained confused for a time and would answer "I don't know" to all questions; but she soon began to brighten up, became interested in work, especially plain sewing, improved physically, and toward the early part of March, 1884, seemed as well as ever. March 10, she was discharged as recovered from the psychosis which had been diagnosed as melancholia.

Soon after discharge a position was secured for her as kitchen helper in a home for incurables at a salary of $7.00 per month with room and board. She has held this position ever since and has rendered highly satisfactory service It is said that she could doubtless get better wages elsewhere, but the conditions being ideal, no change has been made. The patient is even-tempered and has had no trouble with any one at her place of employment. She goes about the city and often visits relatives when off duty. There has been no recurrence of the psychosis and the patient seems exactly as she was before it. Her pleasures are simple, consisting mostly of prayer meetings, church, and visits to relatives. The social worker on this case saw an inscription in a book, which had been given to the patient by the founder of the home at which she is employed, stating that she had been a faithful and valued employee for thirty-five years.

CASE 8.—*Manic-depressive psychosis at the age of 22 years, lasting about seven months; recovery. No recurrence to age 54.*

C. B., a woman aged 22, was admitted May 6, 1889. Her father had a psychosis when young from which he recovered. Otherwise the family history is unimportant. The patient was born in 1876, received a common school education and then went to work as a seamstress. It is said that she had always had good health but was quick tempered. At one time she kept company with a man who eventually married another girl. His marriage occurred shortly before the patient's breakdown and it is thought by relatives that this may have been the cause.

The first evidences of the psychosis appeared about the middle of April, 1889, when the patient began to be sleepless. Her condition grew rapidly worse; she seemed to lose all affection for her parents and brother; she ate poorly, saying the food contained poison; at times she cursed and became violent; she threatened to jump from the window, and had to be committed. The commitment paper states: "Talks and jabbers to herself; sings and shouts; throws her arms wildly about; her answers are irrelevant and contradictory; refuses food and drink until forced; says food is poison; strips off her clothes and would go about in a nude condition if permitted."

On admission she was excited, "running about the halls, regardless of her personal appearance, talking incoherently, and mistaking those about her for

friends long known to her"; she ate poorly. At times she cried freely; said they all wanted to make her marry some one and that they wanted to convert her into the Statue of Liberty; was untidy and often would tear her clothes. Menstruation ceased for several months.

Improvement, both mental and physical, was noted toward the latter part of July, 1889. She became neat in dress, tidy in habits, quiet and rational, and began to take interest in surroundings and people about her; she worked in the sewing room, ate and slept well, and menstruation returned in the latter part of August. She was paroled to relatives Nov. 24, 1889. A week later the father wrote to the hospital stating that she was at times a little cranky, but did not act in any manner out of the way. She was discharged as recovered Jan. 10, 1890.

Since then she has had no recurrence of the psychosis. Relatives objected to our interviewing the patient, fearing that discussion of the attack of mental trouble might upset her. The patient's sister wrote June 14, 1922: "My sister goes to business every day as a first class designer, is enjoying good health and in my opinion has permanently recovered from her mental illness. We could not under any circumstances allow a representative of your hospital to discuss the case with her. This trouble is entirely eliminated from her mind, and we intend at this late date to keep it that way."

COMMENTS AND CONCLUSIONS

The amount of material is small, although our search has been diligent. However, it is not to be concluded from this that permanent recovery from constitutional mental disorders is excessively rare. If it were not so difficult to trace persons thirty or forty years after recovery, especially in such a district as that in which our work was done—a rapidly growing and changing urban district—a great many more cases of such recovery would undoubtedly have been found among the same series of admissions.

Just how often permanent recovery occurs it would be difficult to say. We venture the opinion that in every hospital for mental diseases that possesses records going back far enough to make investigation possible, cases of such recovery could readily be brought to light.

It seems to us that one point is established by our material, limited as it is: current prognostic generalizations concerning recurrence, chronicity, and deterioration in the constitutional psychoses, though no doubt valid for the majority of cases, are not valid for all. Permanent recovery undoubtedly occurs much more often than has been generally supposed. In a given case, especially when the onset is in the second or third decade of life, an unqualified prediction based on such generalizations is not justified.

In the cases here cited it would seem that too large a share in etiology should not be attributed to exogenous factors, though such were present in some. Here, as generally in life, are to be observed new

physical and psychic stresses, sorrows, and disappointments as the years have rolled by without evidence of pathogenic effect. In this respect these cases present a contrast with the general experience of a rise in the incidence of psychoses in correlation with increase in age.

Accordingly, we offer a special relative order of ontogenetic development of the different temperamental elements of personality as an explanation of the unusual course and termination in our cases.

Clinical and Occasional Notes

A TUMOR OF THE CAUDA EQUINA WITH THE FROIN SYNDROME

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Cushing and Ayer recently[1] called attention to the unusual finding of xanthrochromia and increased protein in the spinal fluid above tumors of the cauda equina. This syndrome is most confusing in the localization of cauda equina tumors. That a "Froin syndrome" signifies spinal cord compression, due to tumor or other lesion, sufficiently marked to form a closed cavity and stasis of the fluid in the lumbar culdesac below the lesion, has been generally accepted. Cushing and Ayer report five cases of caudal tumor in which this syndrome was observed—a unique report. The following case report is submitted as another illuminating example:

REPORT OF CASE

Clinical History.—A. B., aged 60, a public accountant, was seen Jan. 12, 1923, because of severe neuralgic attacks along the course of the right anterior crural nerve. The family and personal histories were negative. About August, 1921, he had noticed discomfort and soreness in the inner side of the right thigh, which continued for about six months and then rapidly grew worse. About January, 1922, severe throbbing pains developed in this region, markedly worse in the recumbent posture. This constant pain continued till about July, 1922, when it moderated. Neuralgic attacks, however, occurred in the same region. These were of great severity, extended along the course of the right anterior crural nerve at frequent intervals and lasted about thirty minutes. During the attack he would sit or stand rigidly, unable to move. Large doses of morphin were required to obtain some relief. Since January, 1922, he had been unable to lie down because it would aggravate the pain. During the free intervals he could walk around with a fair degree of comfort. After repeated questioning he stated that at times he felt a slight indefinite numbness along the outer surface of the left thigh. Anal and vesical sphincter control were normal. He had lost 20 pounds (9 kg.) in weight during the year.

Examination.—The results of neurologic examination were negative throughout except for slight irregularity of the left pupil. All reflexes and sensation were normal; there was no evidence of atrophy. Definite trigger areas were found, pressure on which would produce severe neuralgic pains in the course of the right anterior crural nerve. These attacks could be precipitated by: (1) slight pressure on the right anterior crural nerve; (2) deep pressure on the left anterior crural nerve; (3) deep pressure a few centimeters to the right and left of the second, third and fourth lumbar vertebrae. Pressure on the vertebral

1. Cushing, Harvey, and Ayer, J. B.: Xanthochromia and Increased Protein in the Spinal Fluid Above Tumors of the Cauda Equina, Arch. Neurol. & Psychiat. **10**:167 (Aug.) 1923.

prominences elicited no tenderness and no neuralgic attacks; (4) slight stroking of the sole of the right foot, between the ball of the great and little toe.

There was some rigidity of the erector spinae muscles in the lumbar region. Lumbar puncture between the second and third lumbar vertebrae yielded 20 c.c. of spinal fluid under normal pressure, of golden yellow color, which formed a solid coagulum within ten minutes; globulin was in excess; the Wassermann reaction was negative; colloidal gold curve, 0012332110; six cells per cubic millimeter (unsatisfactory because of coagulum). Marked relief of pain was obtained for about twenty-four hours following the spinal drainage. Blood count, blood Wassermann test and urine examination were negative. The roentgenogram of the pelvis and lumbar spine was normal; the roentgenogram of the chest revealed a definite enlargement of the aortic arch. Because of this and the irregular left pupil, antisyphilitic treatment was administered for one month with slight relief of pain. During this time three lumbar punctures were performed with similar spinal fluid findings and temporary relief. Cisterna puncture was advised but not permitted. About this time, February 7, more definite findings developed. Marked edema of both ankles, especially the right, accompanied by profuse localized sweating was observed for a few days; the right knee jerk and achilles jerks were sluggish, the left normal; the right thigh was 3 cm. smaller than the left; indefinite impairment of tactile, pain and temperature sense was present over the left gluteal region. This condition continued and in the absence of spinal cord symptoms, a diagnosis of tumor of the cauda equina was made.

Operation.—The patient was operated on by Dr. William Lerche April 16. A laminectomy, including the arches from the second to the fifth lumbar, was performed. Before the dura was opened a small rounded subdural mass, movable with respiration, was noticed at the level of the fourth lumbar spine. On opening the dura, yellowish spinal fluid escaped both above and below the tumor mass. A tumor about 3 cm. long was found, entirely surrounded by the fibers of the cauda equina. These were dissected off and the tumor was removed without difficulty. The patient awakened from the anesthetic entirely relieved from pain, but complained of marked numbness of the left leg, which subsided after two months. He was discharged from the hospital May 16. By July, recovery was complete; knee and achilles jerks were normal and he resumed his occupation as accountant.

Pathologic Report.—Dr. E. T. Bell, of the Department of Pathology, Medical School, University of Minnesota, submitted the following report: "The tumor is very firm and roughly spherical in shape; diameter 2.5 cm. The cut surface is firm and of a fibrous appearance. There are no areas of softening and no hemorrhages. Microscopic sections show a dense fibrous tissue. The nuclei are spindle shaped, numerous and each is surrounded with a very thin layer of cytoplasm. Between the cells is a large amount of intercellular substance, composed almost entirely of closely packed, fine collagenous fibers. The cells and fibers run in bundles, some being cut longitudinally, others in cross section. No mitotic figures are seen. Diagnosis: "hard fibroma."

Comment.—It might be argued that the Froin syndrome occurring in the spinal fluid above the tumor was due to escape of the fluid from below because of the ball valve-like action of the tumor which was observed before the dura was opened. However, the tumor was not of sufficient size completely to occlude the canal and form a culdesac.

This case illustrates a characteristic of the pains described by Wallgren.[2] He called attention to the fact that in caudal tumors the pain is relieved when the patient is sitting and markedly aggrevated when he is lying down. Our patient was unable to lie in bed for fifteen months prior to operation, because in this position the pain was almost unbearable. Wallgren believes that in the sitting posture the lumbar lordosis is increased, thus shortening the spinal canal and relieving the tension on the sensory fibers in the cauda.

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CONVULSIVE SEIZURES IN SOLDIERS*

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During the past three years I have examined and had under observation 241 ex-service men, beneficiaries of the U. S. Veterans' Bureau, who have been having convulsive attacks due to various causes. Notwithstanding the fact that diagnosis is difficult, I have tried to come to a definite conclusion from an etiologic standpoint in each case; no doubt some mistakes have been made, although, in every case, observation of attacks in hospital has been possible and descriptions have been furnished by a medical officer. Many cases in which the statements of the patient or of others were not verified by such observation in hospital have been disregarded. As will be seen from Table 1, I have diagnosed 40 per cent. of these men as suffering from epilepsy and the balance or 60 per cent., as suffering from various conditions such as congenital inferiority, hysteria, brain injury, syphilis, endocrine disturbance, brain tumor, chronic heart disease, malingering, multiple sclerosis, chronic nephritis and gastro-intestinal disorder, partly manifested by convulsive attacks the cause of which is known. "Meticulous nosology" to quote from Patrick (The Diagnosis of Epilepsy, J. A. M. A. **79**:1009 [Sept. 23] 1922) must therefore be a part of my theme, and I believe it is high time at least to attempt to arrive at some conclusion as to what is and what is not meant by a diagnosis of epilepsy. I have thus confined the use of the term epilepsy, as well as its various derivatives to the syndrome characterizing those attacks that have come to be known as "epileptic fits," but in which the proper pathogenesis is not known with any degree of exactness. I think that the diagnosis of epilepsy should not be used in cases in which identical or similar convulsive attacks occur as part of the symptomatology, but in which the pathogenesis (to include psychogenesis) is well or fairly well known. I classify 60 per cent. of the cases therefore among those convulsive conditions, to which I think the term epileptic should not be

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2. Wallgren: Pain in Tumors of the Cauda, Deutsch. Ztschr. f. Nervenheilk. **78**:107, 1923.

* Read before the Philadelphia Neurological Society, Oct. 26, 1923.

* Publication from the Neuropsychiatric Division of the third district authorized by the Medical Director, U. S. Veterans' Bureau.

applied. Congenital inferiority is placed in this group as it seems to me that there is a peculiar pathogenesis in these cases; namely, a defective anlage, an abiotrophic condition which has resulted in either a mental or physical constitutional defect in structure or in function. The other groups need no explanation. It is of interest to note that of the 40 per cent. of epileptic cases, 23 per cent. existed prior to enlistment and 17 per cent. are thought to have been latent but brought out by war service. While, therefore, war conditions

TABLE 1.—*Percentages of Epilepsy and Convulsive Attacks from Known Causes in 241 Ex-Service Men with Convulsions*

Epilepsy		40
Prior to Enlistment	23	
Service—as precipitating factor	17	
Congenital Inferiority		19
Mental defect	10	
Constitutional Psychopathic Inferiority	9	
Hysteria		16
Brain Injuries		10
Syphilis		6
Congenital	2	
Acquired	4	
Endocrine Disturbances		6
Brain Tumor		0.5
Chronic Heart Disease		0.5
Hystero Malingering		0.5
Asthma		0.5
Multiple Sclerosis		0.5
Chronic Nephritis		0.25
Gastro Intestinal Disturbance		0.25
		100%

TABLE 2.—*Results of Treatment with Phenobarbital*

	Number of Cases			
	Good	Bad	None	Total
Epilepsy	16	2	4	22
Hysteria	2	0	1	3
Trauma	4	0	1	5
Mental Deficiency	1	0	1	2
Constitutional Psychopathic Inferiority	1	0	1	2
Endocrine Disturbance	0	0	3	3
Tumor of Brain	1	0	0	1
Total	25	2	11	38

were probably the precipitating factor in bringing these epilepsies to the surface, they were not the total causative factors. Although only two cases of brain tumor were discovered—one at operation and one at necropsy—it is probable that other cases were not found. In all cases, not only have these men been routinely hospitalized for observation of the convulsive attacks, but, in the great majority of instances, the Wassermann test of the blood serum and of the spinal fluid has been done, as well as roentgen-ray examination of the skull, examination of the eye grounds, and such other special tests as were indicated in individual cases. Of the 241 cases, in thirty-eight records exist as to the

administration and effect of phenobarbital. The dosage has varied between ½ grain (0.03 gm.) and 1½ grain (0.1 gm.), one to three times daily, the maximum daily dose being 4½ grains (0.3 gm.). No deleterious effects of any kind were noted (unless one might term a certain drowsiness, or "dopey" feeling, as the men themselves call it, a toxic result) except in two cases, about 0.5 per cent. of the thirty-eight cases. In these two cases there was a condition of increased irritability and excitability with increased frequency and severity of attacks. There is, therefore, some basis for the opinion that most of the toxic after effects of the drug are consequent on excessive doses. The length of time during which phenobarbital was administered in these cases varied between three months and three years—with an average of ten and one-half months. Results have been recorded as "good" in those cases in which there were fewer and less severe attacks of the same kind, or in which the character of the attacks was modified in a favorable way, as for instance in those cases in which grand mal attacks ceased, and only petit mal attacks continued; or in which attacks of any kind ceased entirely over a long period of time, or in a few instances in which there was only a subjective improvement in general well-being, and an objective improvement as evidenced by less irritability and excitability. The cases included as showing "bad results" were the two already mentioned. Under "no results" were included those cases in which no appreciable effect was obtained. It will be noted that while about 60 per cent. of the cases in which phenobarbital was used were thought to be epileptic, it was also used in a few cases in which the cause of the convulsive attacks was thought to be known. It may then be of interest to call attention to the fact that in three cases thought to be due to some endocrine imbalance, phenobarbital had apparently no effect. The cases thought to be epileptic, and in which good results were obtained, included those marked by either grand mal or petit mal, by both, the so-called mixed type, as well as those marked by psychic equivalents, or by an actual psychosis.

Abstracts from Current Literature

SPONTANEOUS DIFFUSE MENINGEAL HEMORRHAGE. KARL MEYLAHN, Deutsch. Ztschr. f. Nervenheilk. **78**:78 (April) 1923.

Spontaneous hemorrhage of the membranes of the brain and spinal cord, although rare, occurs more frequently than was formerly supposed.

Extradural hemorrhages are usually of minor importance, slight and limited, by reason of the close proximity of the dura to the skull and of the peridural fat and connective tissue to the vertebral canal. Subdural hemorrhages both in the brain and spinal cord frequently are much more expansive and may extend over the entire subdural space. However, definitely defined hematomas occur and are associated with inflammatory conditions of the dura (pachymeningitis hemorrhagica interna). Whether in these cases the inflammatory condition or the hemorrhage is primary is still undetermined. According to Virchow the hemorrhage is secondary to a preexisting inflammatory process. Hemorrhages limited to the subdural region alone are rare, as the subarachnoid space is also usually involved.

Ehrenberg, in 1912, reported two cases in which the hemorrhage was confined to the subarachnoid space and collected twenty-two more cases from the French and Swedish literature. As a rupture can only occur in markedly diseased blood-vessel walls, the author believes that many of these spontaneous diffuse meningeal hemorrhages are due to diapedesis. Cohnheim and Ricker's experiments have demonstrated that a diapedesis hemorrhage can be sufficiently marked to produce grave clinical symptoms and even death.

The reasons that this condition has been reported so rarely are: (1) Frequently these hemorrhages are associated with an organic cerebral or spinal disease which masks the acute syndrome; (2) the hemorrhage may be so slight as to produce very few symptoms; (3) incorrect diagnosis of meningitis, especially in those cases in which a lumbar puncture was omitted.

Luce, Hitzig and others have attempted clinically to differentiate between subdural and subarachnoid hemorrhages. This, as a rule, is not possible. The onset is usually apoplectic in character, soon followed by profound coma, spasticity, contractures, convulsions and frequently exitus. In those cases in which subdural hemorrhage develops slowly and is not extensive, headache and vertigo are the earliest symptoms, gradually followed by coma. If exitus does not occur, the usual syndrome of meningitis develops.

Characteristic also of subdural hemorrhage of the spinal cord is the sudden onset. Excruciating pains and rapidly progressing paralysis without unconsciousness, usher in this condition. Marked rigidity of the spine, severe pains and hyperesthesias due to irritation of the posterior roots are present. The disturbances of motility are either due to spinal cord compression or to the root pains which are aggravated on motion. The deep reflexes are usually decreased. If the hemorrhage is profound, exitus occurs due to shock. Frequently, however, the patient improves with the absorption of the hemorrhage and complete recovery can occur within two months.

At times, prodromal symptoms occur such as hyperesthesia of the spinal column, marked lumbar pains, etc. Rarely the onset is atypical and the usual syndrome of an acute meningitis develops. If the hemorrhage involves the

membranes of the brain and spinal cord, the picture is so confusing that no definite diagnosis can be made.

Lumbar puncture is of the utmost importance. The spinal fluid is under marked pressure and uniformly bloody during the entire drainage. The centrifugated specimen is definitely yellow due to hemolysis.

Spontaneous hemorrhages can only occur from diseased blood-vessel walls, either due to direct involvement or secondary to some disturbance of the vasomotor supply rendering the blood-vessels pervious. There is no seepage from a normal blood-vessel wall even with marked high blood pressure. Syphilis and arteriosclerosis are the most frequent causes for well defined blood-vessel changes. In those cases in which only very slight sclerotic changes occur, occasionally no etiologic factor can be demonstrated. Exciting causes, such as undue emotional or physical strain with a resultant blood pressure increase, are frequently mentioned. Constitutional diseases with a tendency to sclerotic changes are additional etiologic factors. Among these are gout, nephritis, all blood diseases of the anemic and leukemic group, hemorrhagic diathesis, many infectious diseases, chronic intoxicants such as alcohol, nicotin and lead. Cases from the literature are quoted to emphasize some of these etiologic factors.

The author reports two cases with detailed clinical and microscopic findings. The first case was that of a woman, aged 40, in whom marked diffuse hemorrhages occurred from the cerebral and spinal pia and arachnoid. No ruptured blood-vessel was found and the blood-vessel walls were normal throughout. The hemorrhage was supposedly due to diapedesis from the capillaries. No etiologic factor was ascertained and it is believed that the marked diapedesis occurred due to a disturbance of the vasomotor mechanism, at first producing irritation of the constrictors and later paralysis of the dilators, predisposed by an undue constitutional weakness of the vasomotor nervous system. This is in accordance with the theory advanced by Ricker, Dietrich and others.

The second case was that of a woman, aged 53, in whom extensive hemorrhages occurred throughout the subdural region of the spinal cord, most pronounced in the lumbar area. This same condition presented itself subdurally over the entire brain, but to a much less degree. The blood Wassermann reaction was 4 plus positive. Sections revealed marked round cell and lymphocytic infiltration. The causative factor of the hemorrhage was stated to be syphilitic disease of the meningeal blood-vessels. No evidence of a ruptured blood-vessel could be demonstrated.

In his summary the author concludes that spontaneous meningeal hemorrhages are almost always diapedesis hemorrhages. The cause for the production of these hemorrhages is some disturbance in the functions of the vasomotor mechanism. These vasomotor disturbances may be secondary to disease of the blood-vessel walls. In every case, however, it is essential to have a predisposed constitutional weakness of the vasomotor nerves.

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A CONTRIBUTION TO THE PSYCHOLOGY OF APHASIA. C. F. VAN VALKENBURG, Schweiz. Arch. f. Neurol. u. Psychiat. **12**:3, 1923.

Van Valkenburg reports the case of a woman, 39 years of age, who was operated on for an ovarian cyst. Fourteen days thereafter she suddenly lost consciousness for twenty-four hours and on regaining consciousness was completely aphasic. Flames were seen in the right field of vision for one day. Apparently there was a marked right-sided loss of sensation which improved

slowly. She was, at first, able to hear sounds but was unable to interpret what she heard. She became much depressed over a period of eight months, this depression resembling two attacks she had had previously.

The general examination showed nothing abnormal. The results of the aphasia examination were as follows:

1. She was able to imitate simple movements readily. Placing of the hands of a clock or touching the hand to the ear or the eye was, at first, done incorrectly, but later well, although slowly. She was able to copy written and printed words well; she could draw well. Short words could be pronounced easily; longer sentences contained many errors; nonsense syllables were repeated with errors, particularly when the letter combinations were difficult; singing was impossible.

2. Spontaneous movements were well done; writing was fairly well done; drawing was recognizable; recognition of a tapped melody was good.

3. Pointing out certain objects on command was well done. Writing the name of objects seen, heard or touched was fair. Simple colors were easily recognized. Dictation word for word was well done, but longer sentences were poorly done. Spontaneous speech was fairly good. Recitation of memorized verses was impossible; definition and detailed description was poorly done. Supplying the missing words in a sentence was well done. The filling in of picture defects was well done.

4. Reading was satisfactory.

5. General understanding was rather difficult; memory was satisfactory.

6. Reaction to word stimuli was much slowed.

These changes were receptive as well as expressive. It was striking that spontaneous undertakings were carried out in a satisfactory manner. The patient complained that in searching for a word she had difficulty in "hearing this word within herself," that she had to "look into her head as though she were looking into a book," that she had to "draw everything out of the depths." Everything seemed strange as though she had known it, perhaps, a long time before. As soon as the conversation turned to questions and answers the patient began to waver and the words failed to appear and were circumscribed. She could discuss readily and spontaneously a story of her husband's trip across the sea to Scotland, but when asked to do this, and to lay particular stress on the proper sequence of events, the task became very great.

Difficulty in thought is to be distinguished from true aphasia. In this patient there was no failure in thinking; the disturbance of speech did not falsify her thought, nevertheless the mechanism of thought was not normal. The patient complained that one of her chief difficulties was that the thought content disappeared, that words which were used to carry ideas could not be retained. This can be explained by the fact that preceding the internal formation of a sentence, words are used as aids in thinking; words seem to substitute for our thoughts and to serve as props. In the normal individual a word serves as a symbol or a point of crystallization and a substitute for the idea. This was not true in our patient. Here the symbolizing word, in consequence of its indistinctness, represented the entire idea in a broader sense, and a large part of the definition was lost; she was unable to retain several words simultaneously, much less their relation to each other. This accounted for the difficulty in making sentences, indeed she found it easier to write than to speak, inasmuch as she could refresh her memory in this way. An analysis of six pages of her writing showed sixteen mistakes, of which one was purely grammatical, four were amnestic (reduplication of adverbs), six were syntactic, one was the use

of a wrong word, one was the omission of a pronoun, two were paragraphic, and one was paragraphic by anticipation. This analysis showed that the context was disturbed most severely. The sentence rarely broke down at some word that could not be understood. When three sentences, two of which had been falsely constructed, were shown her at the same time the patient was unable to pick out the correct one; however, when these same sentences were shown one at a time the patient had no difficulty in picking out the correct sentence from those that were incorrect. The feeling of strangeness that the patient so frequently referred to is very similar to the phenomenon of déja vu. This was particularly marked in the foreign languages the patient had studied.

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The Pathological Anatomy of the Ductless Glands in a Series of Dementia Praecox Cases. M. E. Morse, J. Neurol. & Psychopath. **4**:1 (May) 1923.

This is the report of a series of carefully planned and controlled studies of the ductless glands, carried out in the Boston Psychopathic Hospital. So much has been said and written in a loose manner concerning these organs that the conclusions of the author are worth quoting verbatim:

The gonads, pituitary, thyroid, and adrenals have been studied in twelve male and fifteen female cases of dementia praecox dying under forty-five years of age. This has been done as far as practicable in connection with control non-psychotic cases of approximately the same ages dying of the same diseases.

An effort has been made to emphasize the sensitiveness with which the ductless glands react to various influences, and consequently the complexity of the problem of interpretation of the changes which are actually found in them in dementia praecox.

In regard to the gonads, the crude results are that sixteen patients had active glands, using as criteria the presence of spermatogenesis and of maturing follicles and corpora lutea. The patients dying of accident or of acute diseases had active glands, with the exception of one case, in which there was only slight spermatogenesis. In tuberculous cases the gonads were atrophic, and the testes showed abnormalities. Two of the women (one originally feebleminded) presented signs of a genital infantilism. Further study of the endocrines in the last two groups of dementia praecox patients, i. e. of the mentally inferior and of those who show stigmata of the hypoplastic constitution, is indicated.

When the tuberculous cases, those developing on a feebleminded or hypoplastic basis, and those dying of wasting diseases are subtracted, the remainder have active gonads.

There was no correlation between atrophy of the sex glands and the duration of the mental disease or the degree of psychic deterioration.

The conditions in the sex glands of the controls were essentially the same as in the dementia praecox cases for the same terminal diseases, with the exceptions of the feebleminded, the infantile and the emaciated cases.

The pituitaries in a little less than half of the cases presented a fibrosis, which could be correlated to some extent with a similar condition in the gonads. As demonstrated by controls, this fibrosis in the pituitary is not peculiar to dementia praecox, but depends rather on the nature and duration of the terminal disease and the state of nutrition.

The lesions in the adrenals were such as are usually found in the diseases to which the patients succumbed.

The thyroids showed changes less frequently than the other endocrines. There was occasionally a mild glandular hyperplasia or increase of connective tissue.

The influence of tuberculosis in producing a fibrous increase in the ductless glands, particularly the gonads and pituitary, has been emphasized once more by the study of our series.

From the pathological side there is little evidence of a primary atrophy of the gonads in dementia praecox, with the possible exception of those cases developing on a basis of mental defect. The fibrosis which is sometimes found in the sex glands is not an isolated change, but is frequently present also in the hypophysis and occasionally in the thyroid. The atrophy, when present, can be accounted for by the somatic diseases from which the patients suffered. This explanation is not only simpler and less hypothetical than that of primary atrophy, but it is more in accord with recent experimental and pathological work on the ductless glands, particularly the gonads.

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FAMILIAL HYPERTROPHIC NEURITIS AND NEUROFIBROMATOSIS. MAX BIELSCHOWSKY, J. f. Psychol. u. Neurol. **29**:182, 1922.

Since Gombault and Mallet first described the clinical picture of familial hypertrophic neuritis, much has been added to our knowledge of this subject. In 1906, Marie described another form of the same disease in which the atrophy was confined chiefly to the lower extremities, while in the upper limbs it was confined especially to the hands. Boveri, who made anatomic studies of one of these cases, found the peripheral nerves to be markedly enlarged, the enlargement being due chiefly to a metamorphic change in the sheath of Schwann. The endoneurium and perineurium showed little change. Like his predecessor, however, Boveri found changes also in the posterior columns of the cord, and to a slight degree in the pyramidal tracts.

Following Boveri's work, Marie and Bertrand added much to the histopathologic picture of hypertrophic neuritis. They found that in the hypertrophic nerves the axis cylinders were not only retained but were increased, the myelin sheath was degenerated and the hypertrophy extended to the most distant portions, the increase in volume being due to hypertrophy of the sheath of Schwann. The cord also showed changes, especially in the posterior columns.

In 1921, Souques and Bertrand studied a sister of a case previously reported by Marie, and found that, while the hypertrophy here was due to changes in the sheath of Schwann, it was more a nuclear than a protoplasmatic change. It was thought that this difference in findings might be accounted for by the stage of the disease process. These authors considered hypertrophic neuritis to be a primary proliferative process of the sheath of Schwann to which later was added a degeneration of the myelin sheath and axis cylinders. It is a "Schwannitis" which involves the peripheral neurons of the motor and sensory tracts in the entire intramedullary and extramedullary extent.

Bielschowsky in his study of hypertrophic neuritis, besides noting the findings referred to, also found signs of nerve regeneration, which was of rather mild degree and of short duration, as the newly formed nerve fibrils soon underwent degeneration. He also called attention to the similarity of the microscopic findings in hypertrophic neuritis and neurofibromatosis. Verocays

has shown that von Recklinghausen's disease presents a similar, though somewhat more variable, pathology. Here the cells of Schwann are the starting points of the tumors, which are rarely entirely nervous, but show admixture with connective tissue.

Antoni has noted three types of tumors: (1) Monocentric, in which the new structure appears to surround the fibers of a nerve bundle and in turn is itself surrounded by nerve fibers which it later invades and produces small nodules; (2) polycentric, in which there are numerous nodule formations, and (3) those in which the tumor produces a sort of jacket about the nerve.

The changes in hypertrophic neuritis are usually of the polycentric type, of which Bielschowsky has seen several cases. In one of these cases two large acoustic tumors were found and also smaller tumors of the other cranial nerves. The spinal nerves appeared normal but microscopically they showed numerous small peritubular tumors.

Several plates are given to show the development of the tumors from the sheath of Schwann. Sections taken from hypertrophic neuritis and von Recklinghausen's disease might be interchanged, so similar are the pictures. The similarity of peritubular neurofibromatosis and hypertrophic neuritis is very evident and can always be established.

Bielschowsky has also seen other types of hypertrophic neuritis in which the hypertrophy was due to a change in the interstitial tissue and not in the parenchyma as is noted in the above two conditions. Other cases are on record in which monocentric tumors have developed so extensively as to produce the picture of a generalized hypertrophic neuritis. Bielschowsky, in calling attention to the close relationship of familial hypertrophic neuritis and neurofibromatosis, questions whether all dystrophic forms of heredodegenerative nervous disease, in which changes occur in the peripheral nervous system, are not closely related to neurofibromatosis, such as the Charcot-Marie amyotrophy, which is identical with the neural form of Hoffman and the Werdnig-Hoffman form. Jendrassik has also called attention to the possible relationship.

It is essential in all cases carefully to examine the skin, especially for nevi, pigmentation disturbances, subcutaneous fibromas and similar anomalies. Such findings might help considerably in establishing a diagnosis, in the same way that sebaceous adenomas do in tuberous sclerosis, which stands in close relationship to von Recklinghausen's disease.

MOERSCH, Rochester, Minn.

THE EARLY SYMPTOMS IN THE DIAGNOSIS OF TUMORS ON THE SPINAL CORD, WITH REMARKS ON THE SURGICAL TREATMENT. CHARLES A. ELSBERG. Am. J. M. Sc. **165**:710 (May) 1923.

The author sums up the progress of spinal cord surgery and points out the frequent mistakes made by surgeons in interpreting abdominal pain; the lesion being an irritation of the posterior roots in the spinal cord rather than from within the abdomen. Pain is an early symptom of spinal cord disease: the pain is persistent in type, strictly localized, and does not radiate for a long time. Later it may be referred to the periphery. In one case reported, the patient had severe pain in the rectum following sneezing, and a lumbar puncture revealed the typical yellowish fluid at the level of the tenth thoracic segment. Pain on sneezing or coughing is often noted. Aneurysm, and disease of the aorta, may give a confusing picture, and must be differentiated from the pain of a true tumor. The author states that pain in the lower part of the back is one of the earliest and most constant symptoms of the large intradural tumors of the

lumbosacral parts of the cord and of the roots of the cauda equina. Tumors with painless onset are of small size, originating from the pia or arachnoid on the anterior or anterolateral aspect of the cord. Early subjective sensory disturbances have been an almost constant finding in his series of cases.

If the growth is situated in the lower cervical and upper thoracic segments, the sequence of the limbs involved as regards motor disturbances is usually: (1) homolateral upper extremity, (2) homolateral lower extremity, (3) contralateral lower extremity, and (4) contralateral upper extremity. Sometimes, however, the contralateral upper limb is affected before the lower limbs, and vice versa. Contralateral motor symptoms, he believes, are due to pressure of the cord against the opposite wall by the tumor. In tumors adherent to the dura, both extradural and intradural, there is often a stage during which the sensory disturbances are more marked on the side of the tumor, while the motor signs are most distinct on the opposite side. This he refers to as the "reverse Brown-Sequard" syndrome. He classifies tumors in their relation to the cord in the following manner: tumors behind the posterior roots are called posterior; those between the posterior roots and the dentate ligaments, are posterolateral; those between the dentate and anterior roots are anterolateral; those in front of the anterior roots are anterior growths.

Of the extramedullary growths, from the author's experience, 62 per cent. were situated on the posterior or posterolateral aspect of the cord, while 36 per cent. were situated anteriorly. The posterior tumors are characterized, above all, by root pains and by very marked posterior root disturbances. Those on the dorsolateral side of the cord are apt to give severe root pains and the Brown-Sequard type of picture.

Tumors on the anterior aspect of the cord very frequently have a painless beginning, with tingling in one or both lower limbs and relatively little sensory disturbance. In posterior, or posterolateral growths, sensory disturbances will usually be found in the peripheral part of the lower extremities.

TEMPLE FAY, Philadelphia.

EXPERIMENTAL RESEARCHES AND CONSIDERATIONS OF CEREBELLAR FUNCTIONS. GILBERTO ROSSI and GINO SIMONELLI, Schweiz. Arch. f. Neurol. u. Psychiat. **12**:24, 1923.

Sherrington introduced the conception of postural tonus, giving us a sufficiently precise definition of that ensemble of phenomena which we recognize under the generic and vague name of muscular tonus. Magnus and his collaborators, in describing a group of reflexes which arise in the labyrinth and in the articulations of the neck, has simplified and rendered more precise the rôle of the physiology of the cerebellum. Luciani's work was of fundamental importance and on it the modern ideas in regard to physiology of tonus have been superimposed. It is evident that we may no longer look on the cerebellum as being responsible for the spasmodic phenomena observed in lesions of the pyramidal tract, if these can be identified with decerebrate rigidity. We must avow that we know little more than hypotheses and phrases concerning the subject of the cerebellum.

The doctrine of cerebellar localization does not yet rest on a secure fundation. Rossi, on removal of the superficial part of the posterior portion of the crus primum, found that the resulting functional disturbances predominated in the upper extremities but also involved the lower extremities, the neck, and

region. These were thought to come from the nuclei cuneati et gracilis. The degenerated corpus restiforme fibers were traced to the caudal portion of the cerebellar cortex.

Section of the lemniscus medialis in the midbrain without injury to the fasciculus longitudinalis medialis, the opposite lemniscus medialis, or the cerebellum resulted in chromatolysis of the cells of all parts of the nucleus tractus solitarii except the commissural part.

GRAY, Chicago.

A CASE OF OCCLUSION OF THE RIGHT POSTERIOR CEREBELLAR ARTERY. D. R. GOWLER and B. M. HOPE, J. Neurol. & Psychopath. **4**:43 (May) 1923.

The lesion was probably embolic from valvular heart disease in a woman aged 53. The onset was sudden with loss of consciousness, on recovering from which there were vomiting, inability to speak and some defect in vision. These symptoms disappeared except that there was still a little hoarseness in speaking. When examined nine months after the onset, the sensory findings were: subjective paresthesia (tingling and burning sensation) of the whole head and of the left side of the body, especially marked about the right eye, the mouth and the tongue; diminution of sensibility to light touch on the left half of the body below the face, with correct localization; reduction of sensitivity and reflex in the right cornea; loss of pain sensibility on the right side of the face and the left side of the body below the face; heat sensibility intact; cold sensibility lost on the left side; tactile discrimination equal on the two sides of the body; deep sensibility unchanged; deep pressure pain diminished on the right half of the face and the left half of the body; diminished vibration sensibility over the whole of the face and head and over the left half of the body; appreciation of weight and stereognosis normal. The sense of taste was lost on the right half of the tongue both back and front.

The right cervical sympathetic was paralysed. No motor paralysis or paresis, and no incoordination were found. In walking the patient tended toward the right, moved slowly, and not without aid. The tendon jerks were moderate, and equal on the two sides. The plantars were of flexor type.

SINGER, Chicago.

LARYNGEAL PARALYSIS ASSOCIATED WITH THE JUGULAR FORAMEN SYNDROME AND OTHER SYNDROMES. GORDON B. NEW, Am. J. M. Sc. **165**:727 (May) 1923.

The author reports seven interesting cases, showing involvement of the ninth, tenth, eleventh and twelfth cranial nerves. In all of the cases, except possibly Case 1, involvement of the nerves was due to neoplasm in the region of the jugular foramen, and even in this case such involvement is the most probable cause, although it is possible the medulla was affected. In six cases the nerve involvement was caused by a tumor. In four of these cases the tumor was slow growing and of a low grade of malignancy; the duration of symptoms ranged from two to twelve years. One lesion of the series was microscopically proved to be carcinoma of the mixed tumor type. Two growths were clinically mixed tumors and one a recurring basal cell epithelioma. In two of the six cases the pathologic lesion was a rapidly growing tumor, probably a lymphosarcoma, the symptoms of which were present for from three to six months only. In these cases the tumor originated in the nasopharynx and pharynx.

In examining the patients, it was noted that those with paralysis of half of the tongue had trouble in swallowing liquids, an act effected by pressing the tongue against the hard palate. Paralysis of the palate caused food to become lodged in the back of the nose. Paralysis of the muscles of the pharynx interfered with the swallowing of solids.

It may be seen from a review of the cases in the literature and from the author's group of cases that laryngeal paralysis associated with a syndrome due to involvement of the last four cranial nerves and the cervical sympathetic nerve in the region of the jugular foramen is quite unusual. When this occurs it is usually due to a neoplasm, although a tuberculous process in the glands of the neck or an acute phlegmon may be the cause. During the World War, cases were reported due to bullet or shrapnel wounds. The vocal cord is in the intermediate or cadaveric position in vagus involvement.

TEMPLE FAY, Philadelphia.

THE PSYCHOBIOLOGIC CONCEPT OF ESSENTIAL EPILEPSY. L. PIERCE CLARK, J. Nerv. & Ment. Dis. **57**:433 (May) 1923.

The author holds: (1) that the concept of an essential epilepsy as a disease entity is justifiable, (2) that various cerebral changes which have been noted are but expressions of an antedating cause and are themselves not etiologically related to the disease, (3) that epilepsy as a disease is dependent on defective functioning of the whole organism. The author stresses the latter view particularly and calls attention to the fact that the epileptic seizure, while the most prominent phase, is but a part of the disorder; all the life activities of the afflicted persons enter the problem and call for the most attentive study. The author notes that deterioration proceeds in many instances in which convulsions have been controlled and that in many cases of epileptic dementia no definite cerebral changes could be demonstrated; accordingly, he believes the interpretation of the deterioration as functional or biologic is justified. The author describes at length the progress of deterioration and finds that prior to the onset of the disease there is evidence of an "epileptic character" in the makeup of the individual. The epileptic character is usually exhibited in the form of "high volatile tempers and all they connote;" in the cases studied this could be demonstrated in all instances.

VONDERAHE, Cincinnati.

PUNCTURE OF THE CISTERNA MAGNA: REPORT ON ONE THOUSAND NINE HUNDRED AND EIGHTY-FIVE PUNCTURES. JAMES B. AYER, J. A. M. A. **81**:358 (Aug. 4) 1923.

It is now somewhat more than three years since Ayer brought into clinical use the puncture of the cisterna magna and described the technic of the procedure. At this time, as a result of a review of his own experience and the reports of a number of physicians, he concludes that cistern puncture, when carefully performed, is a safe procedure; in this series of punctures on 450 patients, no death is known to have occurred directly or indirectly from puncture.

The indications for cistern puncture may be said to be, in the order of importance: (1) in the treatment of meningococcus meningitic block; (2) in the serum treatment of cerebral syphilis; (3) in the early diagnosis of compression of the spinal cord; (4) for obtaining cerebrospinal fluid for examination when elsewhere it is impossible or inadvisable, and (5) as one point of entrance to the subarachnoid space for the purpose of irrigation.

Without a knowledge of the anatomy of the subarachnoid spaces and an appreciation of the physiology of the cerebrospinal fluid, but especially without preliminary experience on the cadaver, cistern puncture should not be undertaken.

NIXON, San Francisco.

ORGANIC NERVOUS DISEASES SUPERVENING IN THE SUBJECTS OF OLD INFANTILE PARALYSIS OR OLD INFANTILE HEMIPLEGIA. F. PARKES WEBER, J. Neurol. & Psychopath. **4**:40 (May) 1923.

After referring to a case he reported in 1899 and to others in the literature, Weber describes two additional examples. In the first patient poliomyelitis occurred at 1½ years and caused atrophy of the right leg with double talipes equinus. At the age of 15, acute drop wrist with atrophy of the intrinsic muscles of the hand occurred first on the left and one month later on the right. The condition improved under treatment and by the end of two months recovery seemed complete. In the second case the poliomyelitis infection occurred at 1⅓ years and caused wasting of the right leg. At the age of 13 and during the four succeeding years there appeared transitory attacks of numbness on one or both sides of the body with the final evolution of a picture of disseminated sclerosis. The only comment offered is that the infantile lesions seem to cause a special liability to disease in the gray and white matter of the cord.

SINGER, Chicago.

SCHIZOPHRENIA AS PSYCHIC SYSTEM DISEASE. KLEIST, Klin. Wchnschr. **2**:962 (May 21) 1923.

In the past twelve years Kleist has gradually come to look on schizophrenia as an endogenous disease. He believes that schizophrenia presents a disease group which may be divided into various subgroups as: 1, the psychomotor form or catatonia, 2, the affective disturbances of hebephrenic type and, 3, the incoherent derangements as schizophrenia in a narrower sense. Closely allied to this would be the schizophasia of Kraepelin and also Kraepelin's paraphrenia.

These disease groups are, according to the author's impression, definite system diseases. They rest probably on the action of endotoxic substances which have an elective affinity for specific systems. They may be looked on as analogous to neuropathic system diseases: types of muscle atrophy, Friedreich's disease, cerebellar atrophies, Huntington's chorea, Wilson's disease, etc. As one may obtain combinations between neurologic system diseases so there may appear combinations of psychic system disturbances.

The fact that various types of schizophrenia may occur in the same family does not indicate a common heredity factor, for in many families only a single form will be observed, while in other families there will be various combinations as with manic depressive types, which are in no way related to schizophrenia.

MOERSCH, Rochester, Minn.

STUDIES OF THE THYROID APPARATUS. XII. THE EFFECT OF THYROPARATHYROIDECTOMY AND PARATHYROIDECTOMY AT 100 DAYS OF AGE ON THE GROWTH OF THE BRAIN AND SPINAL CORD OF MALE AND FEMALE ALBINO RATS. FREDERICK S. HAMMETT, J. Comp. Neurol. **35**:313, 1923.

The effects of thyroparathyroidectomy and parathroidectomy on the growth of the brain and spinal cord were studied in male and female albino rats during the age period from 100 to 150 days. Thyroparathyroidectomy resulted in a

marked retardation of growth of both the brain and spinal cord. The extent of the retardation was greater in the case of the brain in both sexes, and greater for both organs in the female than in the male. Each unit of substance added to the brain of these animals during the growth period was found to contain a much higher absolute amount of solids and a lower absolute amount of water than did the units added to the normal controls. These added solids were also absolutely and proportionately less than those added to the cord of the same animals. Thus the brain seems to be more dependent on the thyroid apparatus for its growth and differentiation than is the cord. Also the growth of the brain is qualitatively and quantitatively altered by loss of thyroid function.

Parathyroidectomy did not disturb the growth ability of the brain and cord to any great extent. The brain was less affected than the cord. Females were more affected than males. Differentiation within the brain and cord did not differ greatly from the controls. It was also observed that the growth of the central nervous system was more resistant to the toxemia following parathyroidectomy than was that of the body as a whole.

GRAY, Chicago.

PRACTICAL MENTAL HYGIENE IN INDUSTRY. F. W. DERSHIMER, J. Indust. Hyg. **5**:1 (May) 1923.

The author, who is not a psychiatrist, recounts his experiences in a group of medical cases among factory employees in which he discovered the predominance of psychogenic factors. His viewpoint of the factory worker is that of the physician rather than the economist: "Too often one thinks of the factory worker as a stolid automaton, unemotionally working out his hours, trudging home to an equally colorless wife, to sup on a smelly stew . . . with never anything approaching thought or emotion." The emotional life of the factory employee, however, the author notes, is extremely varied and complex. With such an understanding of the essentially human qualities of his patients, the author has aided them in forming adjustments to their conflicts with recovery from concomitant physical complaints.

VONDERAHE, Cincinnati.

OSTEOGENETIC DURAL ENDOTHELIOMA. WILDER G. PENFIELD, J. Neurol. & Psychopath. **4**:27 (May) 1923.

The author has investigated 420 case histories of cerebral tumors in the records of the National Hospital, Queen Square, London, and found ten cases with cranial hyperostosis. All proved to be cases of the so-called dural endothelioma (the meningioma of Cushing) with invasion of the overlying bone to form the exostosis. Instead of destroying the bone like most invading neoplasms, it stimulates new bone formation. Brief summaries are given of three cases and the author states that the details will be published elsewhere.

SINGER, Chicago.

THE MENTAL AND PHYSICAL HEALTH OF SCHOOLCHILDREN. SANGER BROWN, 2D, J. A. M. A. **80**:1351 (May 12) 1923.

The observations discussed here are based on a physical and psychologic survey of 150 schoolchildren who were in a special school and of the type often spoken of as difficult or unusual. They are nervous and sensitive; frequently they have unusual temperamental traits. For the children under dis-

cussion the present formal type of education is far from satisfactory. They not only fail to acquire knowledge, but they also get out of sympathy with everything and fail to develop normally.

The particular conditions which the author considers are: (1) nervous instability in children; (2) intelligence tests; (3) physical defects; (4) methods of examination and treatment.

NIXON, San Francisco.

SECONDARY CANCERS OF THE SPINAL CORD WITHOUT OSSEOUS OR MENINGEAL INVOLVEMENT. R. PUECH, Progrès méd. **38**:298 (June 16) 1923.

The secondary cancers of the spinal cord (epitheliomas and sarcomas) are of two orders: (1) In the majority of cases, the cord neoplasm develops by simple extension from tumors of neighboring parts, meninges or vertebrae. In these parts the tumor may be primitive, though more often it is itself secondary to a visceral neoplasm; (2) in other cases, the cord neoplasm results from a metastasis which is directly into the nerve tissue itself, independent of any bony or meningeal participation. Direct metastasis into the cord is infinitely more rare than is direct metastasis into the cerebrum or cerebellum. The writer found only six examples in the literature and reports a seventh.

One conclusion presented is that while secondary cancer of the brain and cerebellum are with great frequency metastatic from breast and lung neoplasm, the secondary cancers of the cord are derived from more varied sites of origin.

DAVIS, New York.

BISMUTH IN THE TREATMENT OF SYPHILIS. J. G. HOPKINS, Arch. Dermat. & Syph. **7**:745 (June) 1923.

Hopkins' study, based on the use of sodium and potassium tartrobismuthate in human patients and in rabbits, confirms the reports of previous observers, who have found in this preparation a powerful antisyphilitic agent. In rabbits, a single injection was found to be about as effective as neo-arsphenamin and more effective than mercuric salicylate. The author feels that with further clinical knowledge of its effects, more favorable results will be obtained by combined treatment with arsphenamin and bismuth, and probably with mercury and bismuth, than by any of these drugs used alone.

VONDERAHE, Cincinnati.

FURTHER NOTES ON THE CENTRAL NERVOUS SYSTEM OF ARACHNIDS: SCORPIONS, PHALANGIDS, AND TRAP DOOR SPIDERS. BERTIL HANSTRÖM, J. Comp. Neurol. **35**:249, 1923.

This is a careful study of the brain of arachnids by the Golgi silver impregnation method. The lateral and medial eye nerves of arachnids enter the optic lobes of the brain. These contain ganglionic masses which consist of a central mass of neuropil surrounded by a layer of small chromatin containing cells. In scorpions there are three such optic masses connected with the lateral eye nerve and two with the median one, but this last mass is common to both nerves. Wolf, crab and wandering spiders have only the first two optic masses connected with the lateral eye nerve. The third mass is connected with the medial nerve which then has the same two masses as the scorpions. In the web-spinning spiders only the first optic mass is retained in the lateral eye nerve;

the number of optic masses in the medial eye nerve remain unchanged. Harvest spiders lack lateral eyes and the masses connected with them, but retain the two optic masses of the medial eye nerve. False scorpions and mites retain only the lateral eyes with a single optic mass, the medial eyes and masses being entirely absent.

GRAY, Chicago.

SOCIETY AND THE CRIMINAL. C. STANFORD READ, J. Neurol. & Psychopath. **4**:35 (May) 1923.

The reactions of society toward the criminal and his treatment are rationalizations and projections of the delinquent cravings of the members of society. For this reason it is the act and not the individual which is the prime psychologic factor, and the crime receives more consideration than the criminal. The unconscious retaliative spirit explains why we have a public prosecutor but no public defender. The attitude of society toward the criminal results in a corresponding feeling on the part of the criminal and recidivism is easily engendered. Society is largely responsible for the behavior problems and should build up its mental hygiene and amend the environmental conditions to produce healthful reactions.

SINGER, Chicago.

UNCOMPENSATED ALKALOSIS IN ENCEPHALITIS. G. A. HARROP and R. F. LOEB, J. A. M. A. **81**:452 (Aug. 11) 1923.

The analysis of the arterial blood in a case of epidemic encephalitis with a disturbance of the respiratory mechanism showed the presence of an abnormally high p_H associated with a low arterial carbon dioxid content, proportionately much lower than the plasma bicarbonate capacity (Van Slyke), which itself is lower than normal. It is evident that the condition is one of "uncompensated carbon dioxid deficit," due to the rapid, shallow breathing, which has produced an abnormally great pulmonary carbon dioxid excretion, with an increase in the ratio $BHCO_3 : H_2CO_3$, and therefore in the p_H.

NIXON, San Francisco.

HERPES ZOSTER GENERALISATUS, REPORT OF A CASE WITH A REVIEW OF THE LITERATURE. M. B. PAROUNAGIAN and H. GOODMAN, Arch. Dermat. & Syph. **7**:429 (April) 1923.

In the case reported here, the patient was a man 64 years of age. A well marked herpes was present in the right abdominal region; in addition, numerous scattered vesicles and ill-defined papules were present on the trunk, on both flanks, axillae, shoulders and arms; the face and buccal mucosa were free from lesions. The probability of herpes zoster with associated varicella was considered. A group of similar cases presenting well marked herpes zoster with a less typical eruption over the body is collected from the literature.

VONDERAHE, Cincinnati.

Society Transactions

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Oct. 26, 1923

C. M. Byrnes, M.D., *President in the Chair*

A CASE OF ENCEPHALITIS PARKINSONIAN SYNDROME SHOWING TRAMPING AND CHAMPING MOVEMENTS. Dr. Temple Fay.

The patient was admitted to the service of Dr. Spiller, at the Philadelphia General Hospital, Aug. 11, 1923, complaining of a "nervous breakdown." She had been perfectly well until eighteen months before when, twelve days after child birth, she noticed drowsiness, salivation and muscular twitchings. A period of insomnia ensued during which she slept little for over two weeks. Recovery from this illness was gradual and uneventful except that she noticed slowness and stiffness in some movements. Friends told her of a change in her facial expression. Four months ago she first noticed champing movements of the jaws. She also developed about this time a slight tremor of the fingers and a continual tramping movement of the feet, appearing as though she were marking time in some definite rhythm. However, the knees are not flexed during this act but the feet are raised from the ground by a partial shifting of weight from one foot to the other.

Physical examination shows a well developed Irish woman, aged about 35, whose responses and sentences are short, and whose face has a mask-like expression. There is a gross tremor of the eyelids when closed and the palpebral fissures are somewhat widened. The sense of smell, the eye grounds and visual fields are normal. The pupils react slowly to light and in accommodation and a slight internal strabismus is present. The facial muscles show slight stiffness in movements innervated by the lower divisions of the seventh nerves. Her mental processes are slow. Hearing is acute and swallowing and speaking are normal. Words are forced out in short, explosive tumults and the inflection of the voice has been lost. The tongue protrudes in the midline and shows many fine tremors along its edges. While the gait is stiff, there is no Romberg sign and the patient finds no difficulty in getting about save for the apparent stiffness of all muscle groups. Reflex responses are normal, except the abdominal reflexes which are lost. Sensation is normal over the entire body and there is no apparent weakness in any muscle groups. Slight tremors are present in the fingers and hands when extended. Passive movements show the muscles to be quite resistant.

The patient is continually moving her jaws as though chewing food or, as she expresses it "chewing gum." The act of swallowing is not associated with this movement. The contraction of the masseter on the two sides is firm and equal, the lower jaw is dropped slightly during each movement; but when the patient is asked to speak or swallow or protrude the tongue, or even when her attention is diverted to some other act, these movements cease.

When the patient stands, she rhythmically sways and lifts her feet slightly from the ground. This movement is controlled at will, and also when sitting, walking or performing some other act. It is not present during sleep.

Conclusion.—The case offers no difficulty in diagnosis—being definitely parkinsonian in type—but there is the unusual appearance of a coordinated associated movement of the jaws and feet. Whether this is a habit or due to definite lesion it seems impossible to determine. If a habit, was there some

irritative cause for its inception If a lesion, it is difficult to explain bilateral associated movements of this character. In some cases there has been demonstrated a unilateral center for bilateral coordinated movements. Champing movements following encephalitis have been noted by several observers. As far as can be determined, the peculiar tramping movements noted in this patient have not been previously recorded.

A CASE PRESENTING PARKINSONIAN SYNDROME WITH CERTAIN FEATURES SUGGESTING PSEUDOBULBAR PALSY, HAVING AN ONSET IMMEDIATELY FOLLOWING CHILDBIRTH. Dr. Gordon F. Willey.

A white married woman, aged 30 years, was second in a family of six children. The heredity and family history are unimportant except that the mother has cancer of the uterus. The birth, infancy, and childhood of the patient are devoid of significant findings. In disposition and in social contacts, including her attitude toward marriage, her reactions have been normal. She has three children, aged 5 and 3 years, and 6 months. A portion of the placenta was retained after the first delivery in 1918, and at this time she was weak, feverish, and ill with influenza from which she recovered in two weeks. At the onset of this so-called influenza there was a sudden sharp pain in one arm and the sensation of being slapped heavily. She was then normal until the birth of her second child in 1920.

One week before the birth of the second child she had what was called "grippe" and was taken with labor pains; immediately after delivery she seemed very ill, had fever, began to sing and talk, was active and apparently delirious. This excitement lasted for two weeks and during this time she was paralyzed in the right side of the face and paretic in the left arm. The face was drawn to the affected side for a year and a half and her speech was indistinct, but the arm recovered in about two weeks when she was able to get up, but complained of pain in the vertex region and rested a great deal of the time. She could not sleep, worried about dying, and cried a great deal. From then on she gradually lost interest, insisted on lying down all the time because she felt weak and tired. She has consulted various physicians and one of them advised her to become pregnant. During pregnancy there was no change in her condition. A child was born in April, 1923, at full term, since when she has been weaker and less active; when sitting up, her head is bent forward and saliva dribbles from the mouth. There has been one menstrual period since the baby was born six months ago.

Physical examination shows apical retraction of the lungs; heart normal; slight abdominal tenderness. The blood pressure is 124 systolic, 88 diastolic. There is a mask-like expression with infrequent winking and swallowing: the body is held stiffly, moved slowly with suppression of automatic associated movements. There is increased tonus of all voluntary musculature with equally increased deep reflexes, more marked in flexors than extensors, but without pyramidal tract signs and abnormal associated movements confirmatory of hemiplegia. The sensory examination is entirely negative, including discriminative touch and the vibratory sense. The pupils are dilated and react widely; the fundi are negative; the voice is whining, and considerable drooling of saliva and slight trophic skin changes are present.

Laboratory Examinations.—The urine is concentrated and shows a trace of indican, and on one occasion traces of both albumin and sugar. The blood count, chemistry and Wassermann reaction are normal. The cerebrospinal fluid was bloody; 278 cells, mainly red cells; globulin increased; mastic

0111111110; Wassermann reaction negative. A roentgenogram of the lower spine shows a defect in union of the lower segment of the sacrum. The basal metabolic rate is normal. Examination of the feces shows occult blood, but no parasitic ova.

Mentally, the patient is clear, cooperative, volunteers but little information, answers briefly and in an indistinct whining tone, at times becoming so emotional as to make speech unintelligible. No delusions, hallucinations, or illusions have been elicited. The mood is depressed with some insight. The patient is quite dependent and appreciative of care. She is correctly oriented and shows an excellent remote and recent memory.

Summary.—A parkinsonian picture, such as is frequently seen after epidemic encephalitis, following childbirth and without a history of encephalitis, with superadded features suggestive of pseudobulbar palsy: namely, weakness of the muscles of the tongue, lips, and palate and to a less extent of the muscles of mastication, not accompanied by fibrillation, atrophy, or the reaction of degeneration; uncontrollable crying in definite attacks, and the low and whining voice. The progress of the disease indicates sequelae of an encephalitis of some description; there has been a degeneration of the cellular elements, particularly the larger, phylogenetically older, cells in the globus pallidus and to a less extent in the putamen with their axons, the so-called paleostriate or pallidal system, a part of the extrapyramidal controlling mechanism. The attacks of apparently uncontrollable crying are similar to those of pseudobulbar palsy and indicate that the paleothalamus had been involved with the paleostriatum.

Several interesting features suggest themselves: The absence of tremor in this case makes it resemble decerebrate rigidity, although tremor is not a necessary part of either the parkinsonian syndrome or its postencephalitic parallel. Tremor of paralysis agitans is probably an inhibitory release phenomenon, while rigidity is an uninhibited tonus. Hughlings Jackson stated in effect that tremor and rigidity may coexist or either may be present without the other, and that their causal mechanism differs not fundamentally, but in degree.

The similarity of this muscular rigidity to catalepsy and catatonic phenomena in general has been suggested, and this problem should permit of solution, but judgment must be suspended as the available literature seems to throw no direct light either on the etiology of the latter or the relation it bears to the parkinsonian syndrome. In any event the constant absence of tremor, the capacity for prompt and complete recovery in the latter conditions, and the chronicity of the former are points against the supposition that the two may be identical.

DISCUSSION

DR. J. HENDRIE LLOYD: Those who have studied the lesions in the postencephalitic parkinsonian syndrome have found them especially in the basal ganglia, midbrain, red nucleus and locus niger. The part of the lenticular nucleus particularly involved is the globus pallidus. In pseudobulbar palsy we have lesions similarly located in the lenticular nuclei. These parkinsonian cases, this case particularly, present pseudobulbar symptoms, such as: paralysis of the tongue, paralysis of the lips, drooling of saliva, and, what is particularly characteristic in some of these cases, explosive emotional conditions, an automatic emotional condition. I see no reason why these symptoms in the parkinsonian syndrome should not be recognized as practically identical with some of the symptoms of pseudobulbar palsy.

I do not know whether my experience has been unusual, but I have seen quite a large group of cases of encephalitis in young married women associated with pregnancy. A recent Italian observer saw fifteen cases of pregnancy associated with encephalitis in an epidemic in Rome. Fifty per cent. of these cases were fatal, but it is a curious fact that the encephalitis seems to have no bad effect on the progress of pregnancy. All the patients whom I have seen went successfully through labor. On the other hand, the termination of pregnancy has no beneficial effect on the encephalitis. It seems incredible that a patient should be advised to become pregnant in this postencephalitic condition, as was done in this case. Two French observers have put on record two cases of pregnancy which occurred in the postencephalitic condition; in both cases there was practically no ill effect on the child, but the effect on the mother was in no respect favorable. These observers say that they could find no similar cases on record. So this case of Dr. Willey's makes the third case on record of pregnancy occurring during the postencephalitic syndrome. In one case reported by Marinesco there was miscarriage and the woman died. In both mother and fetus the evidences were found of epidemic encephalitis.

Dr. William G. Spiller: Some twenty years ago I was impressed by the resemblance of the facies in some cases of pseudobulbar palsy to that of Parkinson's disease, and have still a photograph of a woman taken at that time to illustrate this fact. That was before Parkinson's disease was attributed to a lesion of the lenticular nucleus.

Dr. William B. Cadwalader: During the past year I have seen a number of cases of epidemic encephalitis during pregnancy in Dr. Foulkrod's service at the Presbyterian Hospital. The diagnosis was difficult because of the resemblance to uremia or the intoxication of pregnancy; the question of whether pregnancy should be terminated was difficult to decide.

THE USE OF HYOSCIN IN PARALYSIS AGITANS. Dr. T. H. Weisenburg.

Hyoscin has been used empirically in paralysis agitans for many years. A search into the literature has failed to find the name of the physician who first recommended it. Assuming the pathology of paralysis agitans to be a striatal lesion, the result being an involvement of the involuntary and voluntary musculature, and considering the pharmacology of the hyoscin group, there is evidently no physiologic reason for the use of this drug in paralysis agitans. For over a period of ten years, I have had under observation a number of cases of paralysis agitans. The patients began with the use of $\frac{1}{100}$ grain (0.0006 gm.) of hyoscin once a day. In the course of time they acquired the hyoscin habit to such an extent that one of these patients took thirteen injections each of $\frac{1}{100}$ grain daily.

An investigation as to the benefits derived from the use of hyoscin in cases of paralysis agitans, and in postencephalitic paralysis agitans, has almost invariably shown that patients feel better while under the influence of the drug, because the tremors seem to lessen. On the other hand, investigation of an equal number of patients with paralysis agitans of long standing, who have not had hyoscin, has shown that they are in better condition physically and in addition have the advantage of not forming the hyoscin habit.

I conclude, therefore, that hyoscin is of no value in paralysis agitans or postencephalitic paralysis agitans; and that if it has any benefit, it is because it makes the patient feel better for the time that he is under the influence of the drug, and that no useful purpose can be served by giving this drug in such cases.

MENINGITIC SYMPTOMS RAPIDLY FOLLOWING LUMBAR PUNCTURE AND RAPIDLY DISAPPEARING. Dr. William G. Spiller and Dr. F. L. Payne.

The case was one in which spinal puncture was done within a few hours after the patient had had two epileptic convulsions. Fifteen hours after the puncture the symptoms of meningitis were detected. Twenty hours after the first puncture a second puncture was done. The fluid then obtained was very cloudy, and contained 2,340 polymorphonuclear cells per cubic millimeter, a few mononuclear cells, and a few red blood cells, approximately 100 per cubic millimeter. A culture of the fluid was negative. Forty-two hours after the first puncture and twenty-seven hours after the first clinical signs of meningitis the symptoms of meningitis had about disappeared. (The case will be reported in detail elsewhere.)

The cause of the meningitic symptoms was attributed to a moderate spontaneous meningeal hemorrhage, possibly with irritation therefrom, produced by lowering of the intraspinal pressure after the vessels of the meninges were congested during epileptic convulsions. The fluid obtained at the first puncture showed nothing markedly abnormal.

DISCUSSION

Dr. Daniel J. McCarthy: I have seen in the last year two women with low grade meningomyelitis, with cell counts running from 15 to 18, in whom treatment was changed from the Swift-Ellis method to that of drainage after injection of arsphenamin; after a drainage one of the patients developed promptly a fulminating myelitis of the Landry type.

I think we ought to consider the straight pathology in these cases. We know that inflammation of all tissues of the body may occur that does not go beyond a congestive stage; there is no reason why in a case, like that of Dr. Spiller, of low grade infection with *Staphylococcus aureus* or even with a mild streptococcus there should not have been a first stage meningitis which promtply subsided and then disappeared, just as in the case I am quoting which went on to a very definite inflammatory condition, not only of the meninges but of the cord itself. In his work on experimental meningitis Weed produced a sapremia in cats and then by withdrawing a certain amount of spinal fluid produced definite meningitis. I was inclined to look on my case, in which there was Vincent's angina and pyorrhea, as similar to Weed's cases. I believed we had stirred up not only treponema, but also focal infection and we flooded the cerebrospinal canal with these organisms. I think that would be more rational than to assume a new pathology of out-wandering of leukocytes due to relief of pressure or the presence of minute hemorrhages. I think Weed's work throws a little more light on these cases than we have supposed and that in very many cases we ought to be more careful, when other infections are present in the body, in withdrawing cerebrospinal fluid.

Dr. J. Hendrie Lloyd: I once referred before this Society to a case of a young child with tuberculous meningitis in which a number of lumbar punctures had been made and a serum used as has been recommended by some in tuberculous meningitis. At the necropsy, blood was found extravasated beneath the pia mater. I referred to it as a trauma of the cord due to lumbar puncture, and I can only believe that lumbar puncture is not so simple and harmless as we are led to believe. I have feared at times harm has been done by it. In the delirium of pneumonia, lumbar puncture has been tried, with alleged good results, but it has been suggested that this procedure is not without risk of setting up a pneumococcic meningitis.

DR. WILLIAM G. SPILLER: Spontaneous meningeal hemorrhage has been attracting much attention recently, and papers have been written on this subject by Goldflam, Meylahn and Herman. Such a hemorrhage may give the signs of meningitis. It is true that when a systemic infection exists, lowering the pressure of the spinal fluid is supposed to be capable of causing infection of the spinal cord or its membranes. Physicians are now more cautious in performing lumbar puncture than they were formerly, largely because of the danger of spreading inflammation. In the case we have reported there was no evidence of systemic infection. We are familiar with Weed's work and recognize its importance, but that should not blind us to the importance of other investigations, particularly the recent work by reliable observers on spontaneous meningeal hemorrhage to which too little attention has been paid. This also is "straight" pathology. The possibility of bacterial invasion by lowering the spinal pressure was considered carefully in our case and dismissed. The rapid disappearance of symptoms (twenty-seven hours) could hardly have occurred in bacterial invasion of the meninges. Blood was not found in the fluid obtained at the first puncture but was found in the fluid obtained at the second puncture in the form of a comparatively small number of red blood cells on microscopic examination.

CONVULSIVE SEIZURES IN SOLDIERS. DR. A. J. OSTHEIMER.

This article will be published in full in the ARCHIVES OF NEUROLOGY AND PSYCHIATRY.

EPILEPSY AND MIGRAINE OCCURRING IN THE SAME INDIVIDUAL. DR. N. W. WINKELMAN.

The patient is a married physician, aged 33 years, who complained of attacks of sick headache with all the characteristic eye phenomena of ophthalmic migraine, together with attacks of generalized convulsions with unconsciousness. His family and past histories are unimportant and his habits are excellent. The present illness began about May, 1919, with several attacks of sick headache. He would awaken in the morning with a severe headache and vomiting, which would continue for twenty-four to thirty-six hours. With these headaches he would have eye phenomena which he described as follows: "The eye symptoms take the form of a streak of dancing light, like lightning, which blurs the sight and can be seen with the eyes open or closed. The dancing light comes in from one side, lasts for an hour or more and then breaks up and passes away; recently it has taken another form similar to what one would see looking out on the surface of a clear ocean with a number of black islands."

July 4, 1919, he was found by his family unconscious and in a general convulsion, frothing at the mouth and chewing his tongue. He voided involuntarily and slept for some time; when he awoke he had general muscular soreness. This attack was seen by a physician who made a diagnosis of epilepsy, grand mal.

His blood was negative. His eyes have been examined by Dr. Sweet when the patient was well and also when he was having an attack of migraine. In the former examination the eyegrounds were normal, but during the attack it was found that the left pupil did not react as quickly as the right and that the retinal vessels were very full but did not pulsate. A roentgen-ray examination of the skull showed no pathologic changes.

The case is presented as a combination of epilepsy and migraine. I feel that migraine is a mild type of epilepsy limited to the sensory rather than to the motor sphere; in other words, it is one of the subtypes or one of the manifestations of epilepsy, and as such I am presenting him.

Book Reviews

SUR UN TYPE DE PARAPLÉGIE EN FLEXION D'ORIGINE CÉRÉBRALE AVEC EXAGÉRATION DE L'AUTOMATISME MÉDULLAIRE. By TH. ALAJOUANINE. Volume 5 of Archives françaises de pathologie général et expérimentale et d'anatomie pathologique. Pp. 127. Price, 12 francs. Paris: Gaston Doin, 1923.

In this monograph, Alajouanine reviews an important group of cases in which the position of flexion obtains in the lower extremities. Paraplegia in flexion, he states, was first described by Ollivier d'Angers in 1837 in a case of dorsal Pott's disease. Vulpian, in 1879, and Charcot, in 1893, gave further descriptions of the condition. The cerebral type to which Alajouanine devotes his attention was described clinically by Brissaud, in 1880, by Demange, in 1883, and by Charcot, in 1893. Lhermitte in his monograph on senile paraplegias (1907) described the condition more completely.

Since in these cases the reflexes characteristic of spinal automatism are very evident, Alajouanine has investigated clinically, experimentally and pathologically the characteristics of both the spinal and cerebral form of paraplegia in flexion in order to determine whether a disorder of the same physiologic activities underlies both forms. He concludes that it does.

Alajouanine compares the paraplegia in flexion described by Babinski and due to spinal disease, particularly compression, with the cerebral form which he describes. Clinically, they differ in that there are mental and pseudobulbar symptoms in the cerebral form. Babinski's sign is present in the cerebral form, absent in the spinal form. Involuntary contractions of the legs, sensory disorders and true loss of sphincter control are absent in the cerebral form. Control of urine and feces is often lost in the latter, but this is due to dementia. The upper extremities are not affected. Very marked muscular atrophy with diminished response to electric stimulation, but without reaction of degeneration, is characteristic of the cerebral form. It occurs in patients over 60, while the spinal form occurs at earlier ages as well. In both, the reflexes of spinal automatism are conspicuous.

A case of cerebral origin was described by Marie and Foix in 1920 (*Revue neurologique*, No. 1, 1920). It showed a subependymal degeneration affecting ing the motor fibers arising in the paracentral lobes. Subsequently, Alajouanine examined a case identical from the clinical point of view. At necropsy, he found diffuse degenerations in the cortex, centrum ovale, internal capsule, basal ganglia and cerebellum, but no brainstem or spinal lesions beyond degeneration of the pyramidal and cerebellar tracts. The former he considers characteristic. It is in contrast to the absence of pyramidal degeneration in the spinal form (Babinski). These lesions were of arteriosclerotic origin.

On the clinical side it is interesting, as Alajouanine states, that voluntary power is not abolished, but interfered with by contractures. He believes that muscular power is diminished (possibly due to muscular atrophy—Reviewer).

Alajouanine has investigated the reflexes of spinal automatism in various conditions (fifteen cases) and notes them in unilateral hemiplegias, quadriplegias and striate disorders. He believes that the automatisms of spinal character noted in the cerebral forms of paraplegia in flexion are released by disease of the pyramidal tract as well as of the basal ganglia. He is unable to state with certainty the rôle of the pyramidal tract as contrasted to that of the basal ganglia.

His experimental work showed that bilateral removal of the paracentral lobes in dogs produced the same reflexes of spinal automatism that are found in spinal animals. These persisted for some time after the operation. Unilateral lesions either failed to do this at all or produced but slight manifestations.

Alajouanine finally discusses the various forms of paraplegia in flexion, cerebral and spinal. There are three cerebral varieties: (1) The form he describes; (2) associated with a parkinsonian syndrome due (a) to epidemic encephalitis, (b) to degenerative disease (senile type) or (c) to multiple sclerosis. There is no mental disorder; (3) unassociated with mental changes or pseudobulbar symptoms, due to senile changes in the brain, and known as the myelopathic paraplegia of Demange.

The senile myopathy of Lhermitte, unassociated with clinical or pathologic evidence of disease of the nervous system, due to fibrosis and fatty infiltration of the muscles must be differentiated, since it shows the posture of flexion of the lower limbs. Chronic deforming rheumatism must also be differentiated.

The monograph is interesting, contains a full bibliography and good illustrations.

THE PUBLIC CONSCIENCE. A CASE BOOK IN ETHICS. By GEORGE CLARK COX, New York: Henry Holt & Co., 1922.

This book is the first to apply the case method long used in law and medicine to ethics. In the introduction, Richard C. Cabot expresses the opinion that "Mr. Cox has written an epoch-making book which deserves to alter the tone and method of ethical teaching." From this viewpoint alone the book is worth reading. But it is worth reading because every physician, and the neuro-psychiatrist in particular, is concerned not only with problems of conduct in those whom he has under his care, but also with these same problems in general. Various types of human conduct depend on the more or less complete possession by individuals of adequate health which enables them to react properly to whatever may at any given time be considered the optimum of conduct by the group or state in which they live.

Cox, in discussing the factors influencing conduct, maintains that the theory of the freedom of the will must be set aside "if there is ever to be any progress in moral science." "The theory that by an appeal to the will, or by the Grace of God, the meanest wretch may turn to a life of righteousness and honor, vanishes in thin air."

Cox in treating ethics as a science, in the body of the book, does not concern himself with causes nor with duty and obligation but with a "study of what is, rather than a study of what ought to be." He takes the first step in any scientific enquiry into ethics, namely the discovery and classification of actual cases illustrating various types of conduct in their relation to individuals, groups or states. Though intensely interested in the causes of these varieties of conduct, his book is not a lecture about "oughts" and "ought nots." It may be of interest, however, to know Cox's reply to a request from the late E. E. Southard for a one-sentence summary of his ideas concerning the fundamentals of individual conduct. His reply was "What a man wills to do, modified by what he can't do, is what he ought to do. And there is no other ought." But though this be Cox's idea of individual conduct, it is not inserted into the book, which aims solely to be "a sort of cross-section of contemporary society's judgment about the obligations of its members to the group."

The part which organic diseases of the nervous system plays is emphasized by Cox, and he observes that neuropathologic and psychopathologic investigations have shown that "there are hundreds of thousands of human beings who can by no possibility ever do what is expected of them by society." The neuropsychiatrists who have sufficient initiative to scrutinize diseased individuals not only from the viewpoint of organic or psychic *cause* but also as ethical problems will derive much pleasure and benefit from a study of the technic which Cox has so ably begun for ethics.

The book considers actual court cases involving preservation of life and limb, property, security and liberty. It is pleasantly written, and though erudite in content is by no means labored or difficult in style. Ethics no doubt is identified by many with involved and often dry philosophic discussion. For those who have this idea Cox's book illustrates excellently that works on ethics need be neither obscure in content nor difficult to read.

ESSAI SUR LES CARACTÈRES INTRINSÈQUES DES SECOUSSES MUSCULAIRES ET DES MOUVEMENTS INVOLONTAIRES RHYTHMÉS OBSERVÉS AU COURS DE L'ENCÉPHALITE EPIDÉMIQUE. By EDOUARD KREBS. Pp. 152. Paris: Jouve et Cie, 1922.

Krebs has divided the abnormal muscular movements of epidemic encephalitis into two groups which he compares with types of muscular movement occurring in various diseases. One type of movement is a twitching, a myoclonia, which does not move the limbs or trunk. This is bilaterally symmetrical. The other type of movement is spasmodic and moves the limbs. The movements of athetosis are cited. They are usually unilateral. The first type of movement does not produce deformity of the limbs or trunk, the second finally does. Voluntary innervation stops the first type, but does not stop the second. Krebs states that when no movements are present a "static" contraction will bring on the myoclonic twitchings while a "dynamic" or "kinetic" contraction will bring on the spasmodic movements.

Krebs proposes the following classification of Group 1: A first group of myoclonias, characterized by absence of rhythm and synchronism in the production of the twitchings of different muscles, and by arrest of the phenomena during sleep (paramyoclonus multiplex of Friedreich. . . . myoclonic epilepsy of Unverricht); a second group (this one includes Kreb's cases) which is characterized by rhythm and synchronism of the muscular twitchings, by the absence of the normal muscular synergy which voluntary movement requires, and lastly by the persistence of the contractions during sleep; a third group, characterized by true synergic although jerking movements which cease during sleep (electric chorea of Bergeron and Henoch).

Krebs compares the movements of the second group of motor disturbances, spasmodic in character and very different from the myoclonias, to torticollis, hemiathetosis, chorea and torsion spasms in which, save the rhythm, he finds many of the characteristics of the encephalitic involuntary movements. They disappear during sleep and are increased by emotions and fatigue, which does not occur in the other group. Tonus changes occur in these cases.

The monograph is an interesting discussion of the movements in epidemic encephalitis from a clinical viewpoint.